



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

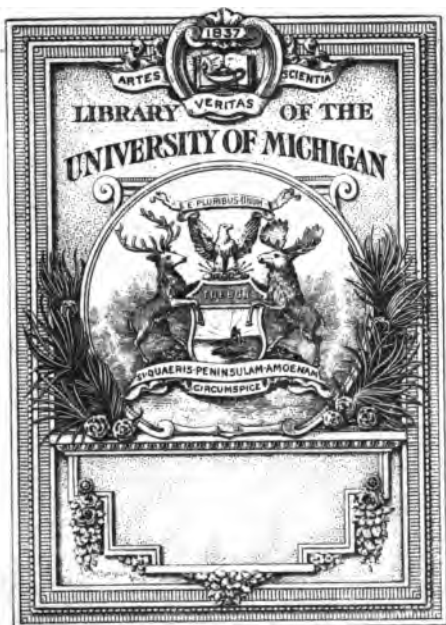
Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

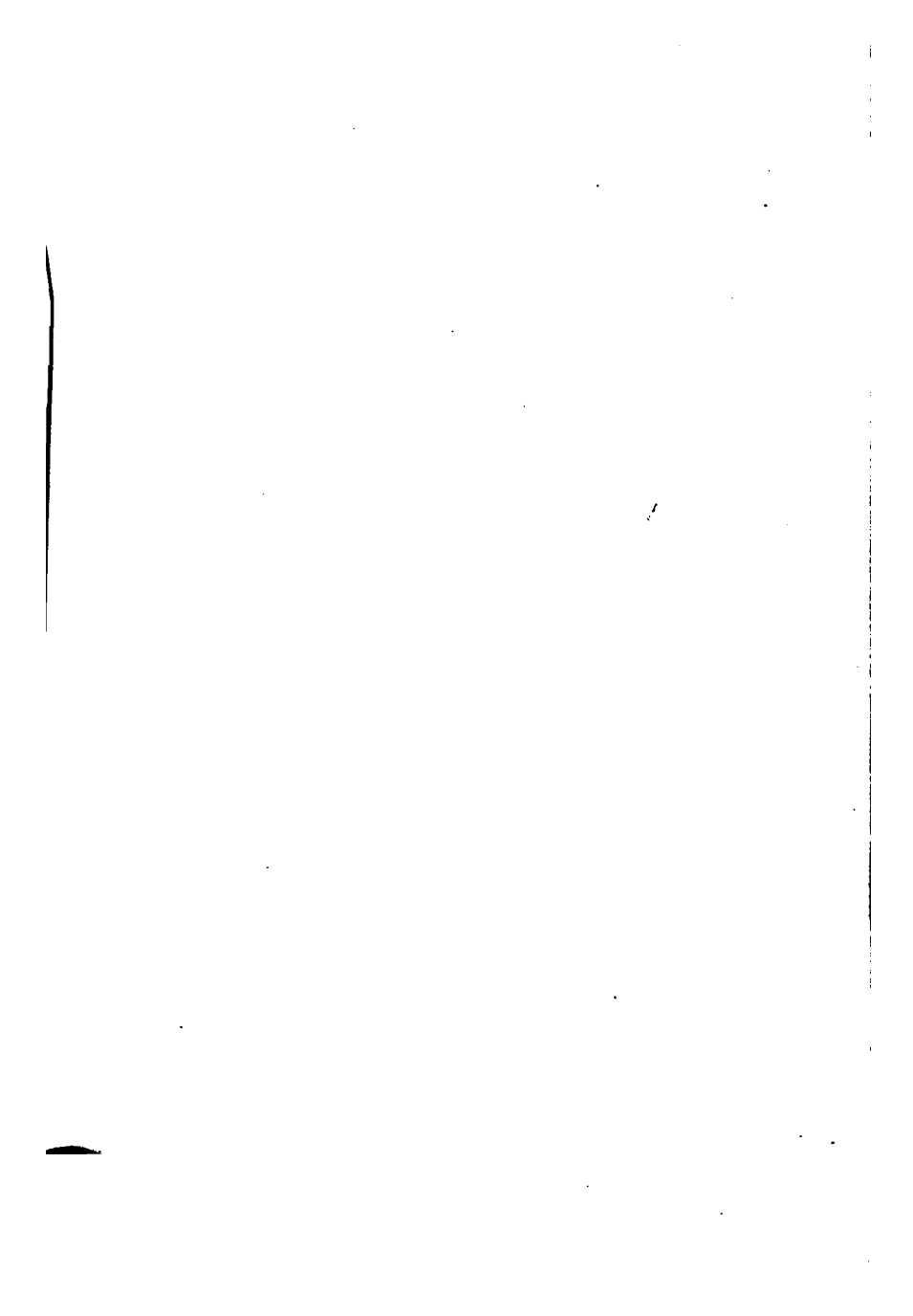
Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



102

H 616.1

H 78



THE HEART AND ITS DISEASES

By
Calvin
C. T. HOOD, M. D.

PROFESSOR OF PRINCIPLES AND PRACTICE OF MEDICINE, CLINICAL
MEDICINE, AND DISEASES OF THE HEART IN THE CHICAGO
HOMEOPATHIC MEDICAL COLLEGE;
ATTENDING PHYSICIAN COOK COUNTY HOSPITAL

PRICE, \$1.00

P. H. MALLEN COMPANY, PUBLISHERS
CHICAGO

COPYRIGHT, 1904,
BY
C. T. HOOD, M. D.

PRESS OF
THE HENRY O. SHEPARD COMPANY
CHICAGO

J 1-18-05-

To the general medical man, who, in his endeavor to meet the demands thrust upon him, has no time for extensive reading, this little volume is dedicated by the

AUTHOR.

130662

PREFACE

“**O**F the making of books upon diseases of the heart truly there is no end.” The object of this small volume is to present to the student and general practitioner what is definitely known of the diseases of the heart and their scientific treatment. Theories and the discussion of the differential diagnoses of heart condition not admitting of a positive diagnosis are omitted.

Each statement as to pathological findings, clinical symptoms and treatment have been repeatedly proven at the postmortem and the bedside, and may be taken as facts.

C. T. HOOD,
1276 Washington boul.

CHICAGO, September 1, 1904.

INTRODUCTION

THE ability to correctly diagnose the ordinary diseases of the heart, and to treat them in the light of modern scientific medical knowledge, can be acquired by any student of medicine. The diagnosis of the less common diseases of the heart is, even in the hands of the specialist, more often speculative than certain. Yet the diagnosis of the ordinary diseases of the heart is, in the majority of cases, a matter of the application of certain well-known laws of physics and the interpretation of long-established clinical data. The scientific treatment of these ordinary diseases of the heart is along lines now past the experimental stage, and is agreed upon by scientific men of all schools; nevertheless, many lives are sacrificed each year from a lack of easily acquired knowledge of the treatment of these diseases on the part of the medical man. There are four principal reasons why the general medical man fails to properly diagnose the ordinary diseases of the heart, and to treat them upon scientific lines:

First. The lack of a proper knowledge of the anatomy and of the physiology of the heart.

Second. The lack of pathological knowledge.

Third. The lack of a scientific knowledge of the action of the so-called heart remedies.

Fourth. But the greatest error is the attempt to study and to treat the diseases of the heart by name, neglecting to study and treat the heart as an organ.

Let us, then, before we take up the study of the

diseases of the heart, spend some time upon the study of its anatomy, physiology and pathology, give a general consideration to the forces concerned in its normal action and the laws of physics under which these forces act, making a review also of the so-called heart remedies.

ANATOMY OF THE HEART

The heart is a hollow muscular organ of conical form, enclosed in the pericardial sac. Its surfaces are generally convex, except where it rests upon the pericardium where that sac comes in contact with the diaphragm and is attached to its central tendon. This portion of the heart is flattened.

The heart is situated obliquely across the median line of the chest, its base up, and beneath the second intercostal space. From the base of the heart arise the large blood vessels *leaving* the heart and which *enter* the large blood vessels going to the heart. These large blood vessels attached to the base of the heart, together with the pericardium, which covers them for some distance, form the only support of the heart, *leaving* it free throughout to move and to enlarge as required.

The shape of the heart varies during life according to its time of action, namely, at rest, filling or contracting, but its vertical measurements do not change. It may be displaced downward by disease.

The dimensions of the heart vary with age, sex, occupation and the general development of the individual. The average measurements of the heart in a well-developed adult male are: length, about 5 inches from base to apex; across the base, $3\frac{1}{2}$ inches; thickness, $2\frac{1}{2}$ inches. As a rule, these measurements increase up to fifty years of age, and decrease after that time of life.

The weight of the heart is about ten ounces in the male, and eight ounces in the female, but the weight will vary to some extent according to the weight of the individual.

When the heart is opened, we find it to be a double *hollow muscular* organ, each side separated from its fellow by a septum, in fact two hearts constructed alike, each able to act as an independent pump. At the base of each of these hearts we find a cavity whose walls are not very thick, each having a small appendix *attached to it*. These chambers are called the auricles or the receiving chambers of the heart.

The left auricle of the heart, which is somewhat smaller than the right, and holds a little less than eight ounces, has emptying into it the pulmonary veins that are returning the blood from the lungs to the heart. These veins are usually four in number — two from the right and two from the left lung. These veins are not guarded by valves — a very important point to remember.

The right auricle has emptying into it the ascending and the descending *venæ cavæ*, the superior *venæ cavæ* having no valves — another important anatomical point to keep in mind. Into the right auricle also empties the coronary sinus that is returning some of the blood from the heart muscle.

At the apices of these two hearts we find two other chambers having thick walls — the left thicker than the right — and taking in the apex of the heart. These chambers are called the ventricles of the heart or the pumps of the heart. All the chambers of the heart and the blood vessels entering the auricles and leaving the ventricles are lined with a tough, smooth, serous membrane called the endocardium.

It will be remembered, then, that the heart is in fact two hearts, each consisting of an auricle or receiving chamber, and a ventricle or working chamber or

pump. Each receiving chamber is connected with the working chamber by an opening, namely, the auriculo-ventricular opening. The opening from the right auricle into the right ventricle is oval in form and large enough to admit three fingers, and is guarded on the ventricular side by a valve usually described as being composed of three flaps. It is, however, not always made of three flaps. This valve is called the tricuspid valve. From the walls of the right ventricle (which, as has been said, are much thicker than those of the auricle) project bands of muscular tissue called the columnæ carneæ. Some of these projections are short and thick, some are long and narrow. The most important of these columnæ are called the muscoli papillares and are three in number, corresponding to the number of the valve flaps of the tricuspid valve, and are well developed. These muscoli papillares are attached by one end to the walls of the ventricle, the other end terminating in a fine tendonous cord that is attached to the valve flap. These are called the chordæ tendineæ. Sometimes these cords are divided into several, before entering the valve flap. They regulate the action of the valve flaps, acting as guy ropes — permitting the valve flaps to close the auriculo-ventricular openings, but not to pass beyond the ventricular side of the opening. These muscoli papillares, as well as the valve flaps, are covered with endocardium that is reflected from the ventricular walls.

The left auriculo-ventricular opening is also oval in shape, somewhat smaller than the right auriculo-ventricular opening, and is guarded by the mitral valve which is composed of two flaps, each attached to a chordæ tendineæ — the termination of a papillary muscle.

Both the right and the left auriculo-ventricular openings are surrounded by a ring of fibrous tissue to which the valve flaps are attached. Under certain circumstances these rings may stretch, enlarging the auriculo-ventricular openings beyond the extent that the valve flaps are able to close, thus permitting the blood to pass from the ventricles into the auricles when the valve flaps are closed.

At the upper part of the right ventricle is the opening of the common pulmonary artery, through which and its branches the blood is forced by the right ventricle to the lungs. The opening into the common pulmonary artery is guarded by three semilunar valves, which are attached to a ring at the beginning of the artery, and are pointed upward, permitting the blood to pass into the pulmonary artery, but preventing its return into the ventricles. At the upper and back part of the left ventricle is the opening of the aorta. This opening is also guarded by three semilunar valves, attached and acting in the same way as those in the right ventricle; only these valve cups are much stronger — made so, to stand the greater strain put upon them. Back of two of these valve cups of the aortic semilunar valves are the openings of the right and left coronary arteries. These arteries are filled when the valve cups are closed, and supply the heart muscle with its arterial blood. The coronary arteries are found in the grooves of the heart between the two sides. The aortic opening is in the groove between the two auricles, and is very close to the left auriculo-ventricular opening. This fact should be borne in mind.

In structure, the heart consists of layers of muscular fibers supported by a framework of fibrous tissue. The

fibrous tissue is arranged in rings about the auriculo-ventricular openings and the aortic and pulmonary openings. To these rings, which are called the skeleton of the heart, are attached the valves and the muscular fibers composing the walls of the cavities of the heart.

The muscular fibers of the heart have a very small amount of connective tissue between them and are of the striated variety. The fibers of the auricles are distinct from those of the ventricles and are composed of two layers, a deep one and a superficial layer. The layers of fibers making up the walls of the ventricles are complicated, and are thicker than those of the auricles, consisting mainly of the so-called "figure-of-eight" fibers, which surround one ventricle and pass on and surround the other ventricle, making contraction of the fibers of one ventricle a contraction of the fibers of the other ventricle.

The nerves of the heart are three in number :

First. Its ganglia — giving to the heart its power to act, or dilate and contract.

Second. The sympathetic — from the cervical sympathetic ganglia — this nerve being the whip or the accelerator nerve of the heart.

Third. The pneumogastric nerve or the inhibitory nerve of the heart.

The heart is enclosed in a sero-fibrous sac called the pericardium, conical in shape, that begins at the blood vessels at the base of the heart and has its base resting upon the diaphragm and attached to the central tendon of the diaphragm. The lining of this sac is reflected upon and covers the heart, and in health

secretes a fluid permitting the heart's surface to move upon or rub the lining of the pericardium without pain.

Facts to remember about the anatomy of the heart :

First. It is a double, hollow, muscular organ, conical in shape.

Second. Its base lies beneath the second intercostal space, one-third to the right of the sternum, and two-thirds to the left of the sternum.

Third. That each heart is practically two hearts.

Fourth. That each of these hearts has an auricle and a ventricle.

Fifth. That the ventricles are larger and their walls much thicker than those of the auricles.

Sixth. That the left ventricle is larger and its walls thicker than the walls of the right, and that the left takes in the apex of the heart.

Seventh. That the right auricle is larger than the left auricle.

Eighth. That the venæ cavæ empty into the right auricle, and that the descending cava has no valves.

Ninth. That the coronary sinus empties into the right auricle.

Tenth. That the pulmonary veins empty into the left auricle, and that they are not guarded by valves.

Eleventh. That the walls of the ventricles are reinforced by muscular columns called the columnæ carneæ.

Twelfth. That there is an opening from each auricle into its corresponding ventricle — the right and left auriculo-ventricular opening.

Thirteenth. That the opening on the right side is guarded by the tricuspid valve, and that the opening on the left side is guarded by the mitral valve, the

flaps of these valves being held in place by the chordæ tendineæ terminations of the muscoli papillares.

Fourteenth. That the cavities of the heart are lined by endocardium and that the valve flaps and the muscular columns are also covered by the same membrane.

Fifteenth. That the auriculo-ventricular openings and the openings of the blood vessels are surrounded by fibrous rings.

Sixteenth. That the common pulmonary artery arises from the right ventricle, and that its opening is guarded by a three-cupped semilunar valve.

Seventeenth. That the aorta arises from the left ventricle, and that its opening is guarded by a three-cupped semilunar valve. Behind two of these cups are the openings of the coronary arteries.

Eighteenth. That the aorta opening is very close to the left auriculo-ventricular opening.

Nineteenth. That the heart consists of a skeleton of fibrous tissue in rings, beginning about the heart openings (b), and of muscular fibers built upon this skeleton of fibrous tissue for the most part arranged in the form of "figure-of-eight" fibers.

Twentieth. That the auricles are connected together and that the ventricles are connected together.

Twenty-first. That the heart has three nerves, namely, the ganglia, the sympathetic and the pneumogastric.

Twenty-second. That the heart is enclosed in a conical sac, the pericardium, the lining of which covers the heart.

Twenty-third. That the supports of the heart are the blood vessels at its base, and the pericardial covering of these vessels.

PHYSIOLOGY OF THE HEART

The instruction on the physiology of the heart as here given may differ in some respects from that taught in the books, but it is the result of much study and experimenting, and if carefully read and considered will very much simplify this important study. We, therefore, commend to the student careful and reiterated reading, knowing that it is impossible to master the pathological conditions of the heart without a thorough knowledge of its physiology.

The circulation of the blood consists of the passing of the arterial or pure blood through the aorta and its branches, from the left ventricle, returning to the right auricle by way of the ascending and descending venæ cavæ, passing through the right auriculo-ventricular opening into the right ventricle, forced by the right ventricle to the lungs by way of the pulmonary artery, returning by the pulmonary veins to the left auricle, passing through the left auriculo-ventricular opening to the left ventricle, to be sent again upon its way.

The mechanism of this action is called the physiology of the heart, which we will proceed to consider.

The time of the heart's action, or the cardiac cycle as it is called, is divided into: First, a time of complete muscular relaxation, or the period of pause of the heart. Second, the time of complete filling of the heart, or the diastole of the heart. Third, the time of the contraction of the heart or the heart's systole.

Let us study these periods one at a time, endeavoring to comprehend the changes occurring each time.

First: the period of rest or pause of the heart.

During this time the walls of the heart are in a state of complete relaxation; the mitral and the tricuspid valves are open; the blood is being forced into the right auricle by the *venæ cavæ*, and is passing through the open right auriculo-ventricular opening into the right ventricle. The blood is being forced into the left auricle by the pulmonary veins, and is passing through the opened left auriculo-ventricular opening into the left ventricle. During this period of the heart's action the semilunar valves that guard the aortic and the pulmonary openings are closed.

Second: As soon as the incoming blood has filled the ventricles, it begins to fill and does fill the auricles. The auricles being filled and the blood still coming into them by way of the *venæ cavæ* on the right side and the pulmonary veins on the left side, the walls of the auricles, being much thinner than those of the ventricles, and unable to withstand the pressure from behind, dilate as far as their muscular fibers will permit and still retain their contractility. Thus put upon a stretch, the muscular walls of the auricles immediately contract, and as at this time the ventricles are filled with blood though their walls are not dilated, the contraction of the auricles forces their contents through the auriculo-ventricular openings. This dilates the ventricular walls, which completes the diastole of the heart.

When the walls of the ventricles have been stretched or dilated by receiving the contents of the auricles, and their muscular fibers having been put upon a stretch, they contract, forcing the blood into the aorta and the pulmonary arteries, distending the coats of these vessels. This is the systole or the contraction of the heart.

As soon as the ventricles cease to contract, and to force blood into the aorta and the pulmonary artery, distending them, the distended muscular walls of the aorta and the pulmonary artery contract. The recoil of the blood closes the semilunar valve cups, closing the openings from the aorta and the pulmonary artery into the ventricles. In the aorta this fills the right and left coronary arteries, the openings of which are behind two of the cups of the aortic semilunar valves.

Let us look at this phenomenon from another direction. During the period of pause, the auricles and the ventricles are in a state of complete muscular relaxation, the auriculo-ventricular valves are open, and the semilunar valves of the aorta and the pulmonary artery are closed. The blood enters the auricles, passes to the ventricles through the open valves, and fills the ventricles, but does not distend their walls. After the ventricles are filled with blood, the auricles are filled with the oncoming blood, then the auricles dilate and contract, forcing their contents into the filled ventricles, dilating the ventricular walls — thus completing the diastole of the heart. As soon as the ventricular walls are dilated, they contract, closing the mitral and the tricuspid valves and opening the semilunar valves, completing the contraction of the heart.

It is to be remembered that the heart has within itself the power to relax and to contract its walls. This can be proven by removing a turtle's heart or a kitten's heart and placing it in a salt solution, when it will be seen to relax and contract. In a strict physiological sense the contraction of the heart begins with the contraction of the auricles, and ends when the ventricles have contracted. The ventricles relax after contracting

and will to a certain extent dilate without receiving the contents of the auricles, but the contraction of the auricles and the forcing of the contents of the auricles into the already filled ventricles completes the dilation. From a clinical standpoint the diastole of the heart ends when the auricles have ceased to contract, and their contents has completely dilated the ventricles. From a clinical standpoint the systole or contraction of the heart ends with the contraction of the ventricles. This point must be borne in mind, as it will help very much to understand the pathological conditions of the heart.

If the ear be placed over the heart near its apex beat, two sounds will be heard, the first a long, deep and booming sound called the first sound of the heart and known as the lubb; the other sound heard is short and snappy and follows immediately after the first sound, and is called the second sound of the heart, or the dub. The first sound of the heart is made at the time when the ventricles are contracting, and is called the systolic sound. In order that we may understand this sound, let us see what is taking place at the time that the ventricles are contracting.

First. The mitral and the tricuspid valves are closed, producing more or less noise.

Second. The semilunar valves are opened, producing more or less noise.

Third. The muscular tissue of the ventricles is contracting, producing more or less noise.

Fourth. The blood is being forced out of the ventricles into the aorta and the pulmonary artery, making more or less noise.

What, then, makes the first sound of the heart?

First. The closing of the mitral and the tricuspid valve flaps.

Second. The "click" of the opening of the semilunar valve cups.

Third. The "sing" of the contracting muscular fibers of the ventricles.

Fourth. The rush of blood as it leaves the ventricles.

The second sound of the heart occurs immediately after the first and at the time of the ventricular relaxation. What is taking place at this time? The muscular walls of the aorta and of the pulmonary artery are contracting, closing the aortic and the pulmonic semilunar valve cups with more or less snap. The second sound is made by the closing of the semilunar valve cups, and is called the diastolic sound, because it follows the systole of the heart, and occurs during the beginning of the relaxation of the heart's muscle.

PATHOLOGY

The endocardium — the lining of the heart which covers the mitral and tricuspid valves and papillary muscles — as a result of certain blood conditions quite frequently becomes inflamed, and from this inflammation the valve flaps themselves become thickened, contracted, glued together or curled up on themselves. At other times, from this inflammation one or more chordæ tendineæ become contracted, or a papillary muscle becomes shortened or stiffened, preventing proper action of the valve flaps. These pathological changes produce two conditions of the valves. First: Imperfect action of the valve flaps from shortening, or imperfect action of the chordæ tendineæ or the papillary muscles. The valve flaps fail to close the auriculo-ventricular openings, permitting the blood to return from the ventricles into the auricles when the ventricles contract. This is a leakage or regurgitation. Second: As a result of inflammation, two or more flaps become glued together, obstructing the passing of the blood from the auricles into the ventricles, causing a stenosis. As a result of disease of the semilunar cups of the aorta, and occasionally of the pulmonary artery, the openings are obstructed and a stenosis results, or they fail to close the opening, and a regurgitation is found. We may have, then, leakage, or regurgitation of the mitral valve, the tricuspid valve, the aortic valve or the pulmonic valve, and we may have obstruction or stenosis at any of the heart valves. These valve changes are called the primary valve diseases. As a result of enlargement or stretching of the heart muscle as a whole, or of one side of the heart, the fibrous rings

that form the auriculo-ventricular openings may become stretched, so that the valve flaps will not close the openings, thus permitting the blood to pass back into the auricles when the ventricles contract. This is what is called a relative leak or a relative regurgitation. As a result of inflammation from the endocardium or from the pericardium, the heart muscle may become diseased or weakened, causing its openings to stretch, and impairing its function. The fibers of the heart muscle may undergo degeneration, or deposits of fat may take place in the muscle or upon the heart itself.

The exact location of the heart valves is of no clinical importance. Experience has proven that there are four areas over which the ear or the stethoscope may be placed and the action heard of the four sets of valves.

First. At the apex of the heart, the normal location for the apex impulse is in the fifth interspace, from one to one and a half inches to the right of the nipple line. In children, the location is often in the fourth space, and in the old sometimes in the sixth space. At this locality, the apex, is heard the closing of the mitral valves, and it is called the mitral area. At the left edge of the sternum or near the ensiform cartilage is heard the tricuspid valve — the tricuspid area. At the second right intercostal space is heard the closing of the aortic semilunar valves — the aortic second area. At the second left intercostal space is heard the closing of the pulmonary semilunar valves — the pulmonic second area. At the apex, two sounds are heard: the first long, loud and booming in character, made by the closing of the mitral valves, and followed immediately by a short, sharp sound, which is due to the closing

of the aortic semilunar valves, called the aortic second at the apex. It will be remembered that the aortic opening is very close to the mitral opening, and this is why the closing of the aortic semilunar valves is heard at the apex. The proof of this is that in certain diseased conditions the second sound is not audible, yet the pulmonic second as heard at the second left space is increased in force. If the second sound as heard at the apex was due in any way to the pulmonic semilunar valves we would hear it at the apex under these conditions.

Diseased conditions of the heart's valves manifest themselves by:

First, New heart sounds called murmurs. There are two kinds of heart murmurs: (a) those due to disease of the heart valves and those due to stretching of the heart's muscle, and (b) those due to other causes than these, the so-called functional heart murmurs. These new heart sounds or murmurs, due to organic changes, may precede, occur with or follow the normal heart sounds. This brings us to the most difficult problem connected with the diagnosis of diseases of the heart, namely, the timing of heart murmurs. New heart sounds that occur at the time that the ventricles are contracting are called systolic murmurs. It will be remembered that at the time that the ventricles are contracting the mitral and the tricuspid valves are closed and the aortic and the pulmonic valves are open, therefore a murmur that occurs at the time that the ventricles are contracting may be (a) at the mitral valve, in which case the blood is passing through an imperfectly closed mitral valve — one whose valve flaps are so altered by disease themselves, or by disease of

their chordæ tendineæ or **papillary** muscle, that the auriculo-ventricular opening is imperfectly **closed** by the valve flaps, permitting the blood to pass back into the left auricle when the ventricle contracts, causing a leakage or regurgitation; or else (b) from disease or stretching of the fibrous rings of the heart — those that make the auriculo-ventricular openings — the opening is enlarged so that the valve flaps can not properly close the enlarged opening, and as a result there is a leakage back into the auricle, or regurgitation occurs when the ventricle contracts — a relative mitral leak giving a relative mitral systolic murmur.

Second. A murmur may occur at the tricuspid valve from the same causes, giving a tricuspid regurgitation either primary or relative.

Third. There may be some defect of the aortic or of the pulmonic valve, preventing the blood from passing out of the ventricles in a proper manner. As a result, a murmur is heard at the second right intercostal space, or at the second left intercostal space with the contraction of the ventricles a systolic murmur is heard, and is either an aortic or a pulmonic obstructive murmur or stenosis.

Fourth. If there be a defect in the mitral or the tricuspid valves so that the valve flaps are glued together, obstructing either the mitral or the tricuspid openings, preventing the blood from passing in a normal manner from the auricles into the ventricles, a murmur will result in most cases. It will be remembered that the blood is passing from the auricles from the time that the ventricles cease to contract and their walls to relax, opening the mitral and the tricuspid valves until the ventricles again contract, but that the blood

is passing with greater force from the auricles into the ventricles at the time that the auricles are contracting. This contraction of the auricles distends the ventricles and immediately precedes the contraction of the ventricles; therefore, if a murmur is to occur at any time preceding the contraction of the ventricles at the mitral valve or the tricuspid valve, it will occur at the time that the auricles are contracting, or just before the systole of the ventricle. Such a murmur is called a presystolic murmur and means obstruction or stenosis of the mitral or tricuspid valves. It will be remembered when the ventricles contract, throwing their contents into the aorta and the pulmonary artery, the muscular walls of the aorta and of the pulmonary artery are dilated or stretched, and as soon as the ventricles cease to contract that the elasticity of the muscular coats of the aorta and of the pulmonary artery contract and close the aortic and the pulmonic semilunar valve cups with a snap. If there be any disease of the aortic or pulmonic semilunar valves preventing their properly closing the aortic and the pulmonic openings, the blood will be forced by the contraction of the muscular coats of these vessels back into the ventricles — an aortic or a pulmonic regurgitation, and as at the time that the aortic and the pulmonic valves are closing the ventricles are preparing to fill and dilate, a murmur that is heard at this time is called a diastolic murmur. We may have, then:

First. A systolic murmur at the mitral valve, either primary or relative, a mitral regurgitation.

Second. A systolic murmur at the tricuspid valve, either primary or relative — a tricuspid regurgitation.

Third. A systolic murmur at the aortic valve — an aortic stenosis.

Fourth. A systolic murmur at the pulmonic valve — a pulmonary stenosis.

Fifth. A presystolic murmur at the mitral valve — a mitral stenosis.

Sixth. A presystolic murmur at the tricuspid valve — a tricuspid stenosis.

Seventh. A diastolic murmur at the aortic valve — an aortic regurgitation, which is always primary, that is to say, while the mitral and the tricuspid openings may become stretched by disease so as to produce a relative regurgitation of those valves, the aortic valve opening, or the rings that form them, never become so stretched that a relative leak or regurgitation of the aortic valve or its rings, or of the pulmonic valve, occurs.

Eighth. A diastolic murmur at the pulmonic valve — a pulmonic regurgitation, which, however, is a very rare condition.

These are all the heart murmurs that may occur of organic origin. The second manifestation of diseased valves of the heart are changes in the heart muscle, either (a) enlargement of the heart as a whole or in part, or (b) dilation of the heart muscle either as a whole or in part. The third manifestation of disease of the valves of the heart is disorder in the systemic and the pulmonic circulations. The fourth manifestation of the diseased valves of the heart are certain clinical symptoms.

There are certain things about organic heart murmur that must be distinctly remembered:

First. The time of the murmur, whether systolic, presystolic or diastolic, that is, occurring with the contraction of the ventricle; systolic, or occurring before

the contraction of the ventricle ; presystolic, or occurring when the aortic or the pulmonic semilunar valves close (at this time the ventricles are dilating) — a diastolic murmur.

Second. The place on the chest where the murmur reaches its greatest intensity — where we hear it best.

Third. The direction in which the murmur is conducted, whether up into the vessels of the neck, or down toward the apex or along the left edge of the sternum or to the right or to the left.

Most writers on this subject enumerate several other points to be remembered, but these are the ones upon which the most stress is placed.

With this study of the anatomy, physiology and pathology of the heart, let us proceed to the study of individual heart lesions. We trust that the student has made himself familiar with the subjects that have been gone over ; for, if he has not, the study of the diseases of the heart will be a very difficult matter ; in fact, we doubt if, without this familiarity, anything like a positive knowledge of the lesions of the heart can be acquired, and, indeed, it is for this reason that so many otherwise good general medical men are unable to diagnose the ordinary diseases of the heart.

Most writers on the heart take up for consideration, first, diseases of the pericardium, then those of the myocardium or heart muscle, then those of the endocardium ; but as the proper understanding of these pathological conditions depends to a large extent upon the understanding of the valvular defects, we shall consider those pathological changes and their symptoms first.

MITRAL REGURGITATION

Mitral regurgitation is the most common valvular defect found in the heart. It is one that every medical man will be called upon to diagnose and treat, and the proper understanding of mitral regurgitation will be of great help in the understanding of the other heart lesions. If we spend some time in its consideration, we may be satisfied that the time will be well spent.

Mitral regurgitation may be of three kinds. First: Acute mitral regurgitation due to acute dilation of the heart muscle, stretching of the mitral orifice and permitting the blood to pass back into the left auricle when the ventricle contracts. In this case, all the heart valves may be leaking, and either death result or the heart muscle slowly takes up, closing the stretched openings. Second: Relative mitral regurgitation, due to enlargement or dilation of the left ventricle, this enlargement being the result of hypertrophy or disease of its walls. The enlargement or dilation stretches the mitral orifice beyond what the mitral flaps are able to close, permitting the blood to pass back into the auricle when the ventricle contracts. Third: Primary organic mitral regurgitation, due to disease of the valve flaps — the chordæ tendineæ or the papillary muscles.

ETIOLOGY

Primary mitral regurgitation is due to but one cause, namely, endocarditis. This endocarditis may be acute or chronic and involve the endocardial covering of the valve flaps, the endocardial covering of the papillary muscles, and to a greater or less extent the endocardial lining of the ventricle. In the greater number

of cases it is the endocardium covering the valve flaps, or that covering the valve flaps and an inflammation of the chordæ tendineæ, or the covering of the valve flaps and the papillary muscles and an inflammation of the chordæ tendineæ, but more often it is the valve flaps alone that are involved in the inflammation, or, in other words, the disease is a valvulitis. As a result of this endocarditis or valvulitis, the valve flaps become distorted, their edges roughened, curled, twisted or contracted, or the chordæ tendineæ are contracted from inflammation, producing unequal tension upon the valve flaps; or the papillary muscles are contracted, making unequal tension upon the valve flaps. Again, these papillary muscles become stiffened or adherent to the sides of the ventricle, preventing proper action of the valve flaps. But, be the pathological changes what they may, the result is the same, namely, the imperfect closing of the auriculo-ventricular openings, permitting the blood to return to the auricle when the ventricle contracts.

It will be remembered that diseases of the heart valves manifest themselves by: First, murmurs. Second, changes in the heart muscle. Third, change in the systemic and the pulmonic circulations. Fourth, clinical symptoms.

Let us reverse the manner of study, and consider first the changes in the heart's muscle and in the systemic and the pulmonic circulations. We know from the study of the anatomy of the heart that in a normal adult male heart the left ventricle holds about eight ounces of blood. If the mitral valve be imperfect, and the valve flaps, from any of the diseased conditions that have been mentioned, fail to close the mitral open-

ing when the ventricle contracts, a leakage or regurgitation of blood will take place into the auricle. Let us say that this regurgitation is two ounces, that is, each time that the ventricle contracts there will be two ounces of blood forced back into the auricle by the contraction of the ventricle. Two things, then, must take place: First, the aorta, that in health receives the contents of the left ventricle—eight ounces—will receive eight ounces, less the two ounces that is forced back into the auricle through the defective mitral valve by the contraction of the ventricle; and, second, the left auricle will have crowded into it two ounces more of blood than in health. In order that the aorta may receive its normal amount of blood—eight ounces—each time that the left ventricle contracts, the left ventricle must contain at the time of contraction eight ounces of blood (the normal amount), and two ounces (the amount leaked back into the auricle), and in order that the left auricle may be able to contain this extra two ounces its walls must dilate to hold it. In order that the left auricle may be able to force this extra two ounces into the left ventricle, its walls must become stronger or, in other words, extra work being thrown upon the left auricular walls means hypertrophy of the walls of the left auricle. Again, in order that the left ventricle may be able to hold the extra two ounces of blood, its walls must dilate to receive it, and its capacity must be increased two ounces. In order, therefore, that the left ventricle may be able to force eight ounces (the normal amount of blood) into the aorta, and two ounces (the amount of the leakage back into the left auricle through the defective mitral valve), its walls must become stronger or they must hyper-

trophy. These are the changes that take place in the greater number of cases, in fact in all cases. The leakage is at first very slight, so that the enlargement and hypertrophy of the left auricle and of the left ventricle take place very gradually and go synchronously; at other times the leak is extensive, and it requires some weeks for these changes to take place.

As long as the leak is slight and the left auricle is able to receive the extra amount of blood from the left ventricle through the defective mitral valve when the ventricle contracts, and the auricle is able to enlarge sufficiently to hold and to force into the ventricle the extra blood, no other changes occur, and no symptoms result except a systolic murmur heard at the apex. This murmur is transmitted to the left and is often heard in the back, and, with some slight signs of enlargement of the left side of the heart, are all the evidence we have of disease.

But such cases as these are the rare ones, and in the greater number of the cases other changes take place as follows. It will be remembered from our study of the anatomy of the heart, that the pulmonary veins which return the blood from the lungs to the left auricle have no valves, and that the blood is held in them by the blood pressure in the vessels of the lungs. Keeping this fact in mind, it will be easy to understand why, when the left auricle has dilated and hypertrophied to the fullest extent possible in order that it may hold the blood forced back into it through the defective mitral valve, and the auricle is no longer able to withstand the strain put upon it by the blood pressure within it, that the blood is dammed back in the pulmonary veins, into the pulmonary circulation.

and as time goes on and the leak at the mitral valve increases, and more and more blood is forced into the left auricle by the left ventricle through the increasingly defective valve, the blood becomes dammed back not only into the pulmonary veins and their branches but through the pulmonary capillaries into the branches of the pulmonary artery, and finally back through the pulmonary artery to the right ventricle. This increase in the blood pressure in the pulmonary circulation produces congestion of the pulmonary capillaries, and the water of the blood is squeezed out of the capillary walls in the form of an exudate into the air cells and the smaller bronchial tubes, and not infrequently a small capillary is ruptured and blood escapes and is expectorated with the mucus. Cough is one of the first and most prominent symptoms of this backward blood pressure in the lungs, from leakage of the mitral valve. With this cough there is more or less dyspnea, which is manifest most upon exertion, such as running, or climbing stairs, or extra lifting. As soon as the increased blood pressure has reached the right ventricle, by way of the pulmonary artery, more work is thrown upon it. To overcome this increased blood pressure in the pulmonary artery and its branches, the pulmonary capillaries and the pulmonary veins, and to assist the left auricle in its efforts to fill the left ventricle, this extra work put upon the right ventricle requires that the walls of the right ventricle must increase in size to be able to meet this extra demand put upon it, therefore its walls increase in size or hypertrophy. As soon as the walls of the right ventricle have hypertrophied sufficiently to be able to overcome the back blood pressure in the pulmonary

circulation, and be able to assist the left auricle in its work of filling the left ventricle, so that the left ventricle receives enough blood when it contracts to throw a normal amount in the aorta and at the same time leak into the left auricle through the defective mitral valve, what is called compensation has taken place. Or, in other words, a leak at the mitral valve dams the blood back through the pulmonary circulation into the right ventricle, which must increase its working power sufficient to overcome it and help the left auricle with its work. That is to say, that when the mitral valve leaks to any extent, the work of overcoming the results of that mitral leak falls upon the right ventricle, or a mitral leak is compensated for by hypertrophy of the right ventricle. Knowing, then, the changes that result from mitral regurgitation, how are we to know that these changes have taken place in a given case? How are we to know that the right ventricle has enlarged or hypertrophied? This brings us to a much disputed point, namely, the percussion of the heart. We are well aware that most of the writers on this subject spend considerable time upon directions for percussion of the heart, but we are also aware that except in the hands of a very few experts the percussion of the heart yields no information whatever, and to the general medical man is useless, and a waste of time that had better be spent upon other and more important points. That the heart has a deep and superficial dulness we are aware, but so many things may influence it that we shall eliminate percussion altogether from our study, and endeavor to spend the time upon points of greater simplicity and accuracy. To return to our subject — how are we to know that the right

ventricle is enlarged or hypertrophied? The normal location of the apex beat is in the fifth intercostal space $1\frac{1}{2}$ inches to the right of the nipple line. When the right heart, that is, principally the right ventricle, is hypertrophied the apex will be found in the fifth interspace, or a little lower, say under the sixth rib, but the apex will be to the left of the normal line from $\frac{1}{2}$ to 2 or 3 inches. That is to say, when the right ventricle is enlarged or hypertrophied the apex impulse will be misplaced to the left, often a little down but much more to the left than down.

The aortic second sound, as heard at the second right intercostal space, depends upon: First: The amount of blood thrown into the aorta. Second: The force with which this blood is thrown into the aorta. Third: The pressure of the blood in the aorta and its branches, or the blood pressure in the systemic circulation. Fourth: The contraction of the muscular coats of the aorta. The contraction of the muscular coats of the aorta will depend upon whether these coats are diseased or not.

The snap with which the pulmonic semilunar valves close as heard at the second left intercostal space, will depend: First: Upon the amount of blood thrown by the right ventricle into the pulmonary artery. Second: The force with which the blood is thrown into the pulmonary artery. Third: The blood pressure in the pulmonary circulation. Fourth: The contraction of the muscular coats of the pulmonary artery, and the amount of contraction of the coats of the pulmonary artery will depend upon the condition of its coats.

If, as is the case in mitral regurgitation, the blood pressure in the pulmonary circulation is high, or is

increased from the damming back through the defective mitral valve, and the coats of the pulmonary artery are not diseased — which they rarely are — and we listen at the second left intercostal space, we will hear the pulmonic valves shut with a distinct snap. From this we may judge, other things being considered that will be spoken of later, that the right ventricle is throwing an abnormal amount of blood into the pulmonary artery and with an abnormal force. If we find that the apex beat is of a good forcible character, in the fifth intercostal space or under the sixth rib from one to two inches to the left of the normal line, and if, upon listening, we find the pulmonic second sound as heard at the second left interspace, called the pulmonic second, is accentuated, we may judge that the right ventricle is hypertrophied.

Given, then, a case of mitral regurgitation, in which the leak is compensated by hypertrophy of the right ventricle, upon what shall a diagnosis be made and what will be the symptoms?

First. The pulse will be regular (it may be very irregular), quite full and of moderate tension.

Second. The apex beat will be seen and felt in the fifth interspace or under the sixth rib from one to three inches to the left of the normal line.

Third. The impulse will, as a rule, be regular, distinct, not a tap and not of a heaving character.

Fourth. Upon auscultation at the mitral area, the apex, a soft, blowing, fairly loud, often musical murmur, systolic in time, is heard. This murmur is transmitted to the left and is heard in the mid axillary line and under the angle of the scapula behind, and is often heard to the right of the apex and up toward the base,

gradually decreasing in volume the further away from the apex one listens.

Fifth. At the second left intercostal space, the pulmonic second sound will be heard as a distinct snap, or it is accentuated. [NOTE.—During youth and up to twenty-six years of age, the pulmonic second as heard at the second left space is more distinct than the aortic second as heard at the right second space. After twenty-six years of age, and up to fifty or fifty-five years of age, the sounds are about equal, the pulmonic second being a little more distinct during the first part of this period, and the aortic second more distinct during the latter part, and after this age the aortic second becoming more and more distinct. These facts must be taken into consideration in forming a judgment as to whether or not the pulmonic and aortic seconds are normal, below normal or accentuated.] Dyspnea upon exertion will be the most important symptom. Cough, which is very much increased upon taking a little cold or walking against the wind, some expectoration of a white frothy nature often streaked with blood, but no distinct hemorrhage. Palpitation of the heart is quite common — especially is this true when stomach digestion is not good and gas is present in the stomach or even the bowels, making pressure upon the under surface of the diaphragm and impeding the heart action. But so long as no extra strain is put upon the right ventricle, the patient can go along about the ordinary duties of life with comparative comfort, run no risk in taking an anesthetic, bear children and perform some manual labor.

Treatment of Compensated Mitral Regurgitation

Well compensated mitral regurgitation — that is, one in which the right ventricle is able to maintain the pulmonic circulation at about a normal height and assist the left auricle in filling the left ventricle, so that the left ventricle may throw a normal amount of blood into the aorta and still leak at the mitral valve — such a case requires but little treatment, and many such cases go for years undiscovered. People suffering with a well compensated mitral regurgitation should be told to avoid extra exertion, as running, going up stairs in a hurry, lifting, over excitement of any kind, taking cold, indigestion which results in gas and a distended stomach. Moderate exercise, as deliberate walking, riding, etc., will do good; even mountain climbing is beneficial if done in such a way as not to tax the heart. If the general health becomes impaired from any cause, and the heart muscle suffers in the general let-down condition of the muscular system, such drugs as nux vomica and iod. of arsenic will in all probability be all that is necessary, with some attention to diet and rest till the heart muscle has caught up. But if such a patient contract a severe cold and have a bronchitis, or overdo in any way so as to put extra strain upon the right ventricle, by degrees the fibrous bands that make up the tricuspid openings stretch until the opening becomes enlarged beyond what the tricuspid valves are able to close, and as a result each time that the right ventricle contracts some blood is forced back into the right auricle — a relative tricuspid regurgitation. The tricuspid opening continues to enlarge, and more and

more blood is forced into the right auricle until the blood pressure in the pulmonary artery and the pulmonary circulation is decreased because the right ventricle is not throwing the usual amount of blood into the pulmonary artery. The lungs become more congested from the dammed back blood from the left side of the heart — the mitral leak — the cough is more severe, the dyspnea more and more increased; the amount of blood forced by the left auricle into the left ventricle is less; the amount of blood forced by the left ventricle into the aorta and the systemic circulation is less; the blood pressure is lowered in both the pulmonary and systemic circulations. The pulse becomes small and irregular, the nails blue, the breathing more or less labored, the expectoration markedly increased and more or less bloody. As a result of this leak or regurgitation of the tricuspid valve, from stretching of its fibrous rings, not from disease of the valves, a relative regurgitation, the blood is dammed back into the right auricle, and as the right auricle is still receiving blood from the ascending and descending venæ cavæ, the right auricle becomes overfilled, and its walls, not being able to stand the force in front, the contraction of the right ventricle, and the force behind — the blood pressure in the venæ cavæ — the auricular walls dilate, and to a very slight extent hypertrophy, but not sufficiently to overcome the strain put upon them. The blood is then dammed back into the venæ cavæ, and as the descending cavæ has no valves, the pressure is first felt in this vein and its branches, the jugulars. Each time that the right ventricle contracts and forces blood through the stretched tricuspid orifice into the right auricle, the jugulars will be seen to pulsate, and as the

tricuspid leak becomes greater the ascending venæ cavæ feels the back pressure and the blood becomes dammed back into this vessel and its branches. The liver becomes congested or engorged with venous blood; the veins that go to make up the portal vein become engorged; the liver will pulsate with each contraction of the right ventricle; catarrhal conditions of the stomach and bowels result from transudation of blood serum from the overfilled veins. The kidneys become passively congested; the amount of urine is decreased, becomes turbid and loaded with urates, and may contain a small amount of albumen. After a time, varying in length, depending upon the rapidity with which the tricuspid orifice stretches, the venous pressure in the legs and feet increases and the arterial pressure diminishes, due to lack of proper amount of blood in the left ventricle. The capillaries of the extremities become congested, and this is most marked in the feet, the most dependent portion of the body — the blood serum exudates from the capillaries into the lymph spaces of the tissue, and dropsy, or anasarca, begins. This dropsy first involves the feet, then the ankles, the legs, then the hips and back. When the pressure becomes so great upon the portal veins and the portal circulation that it can not stand it, the blood serum is transuded into the abdomen, and ascites results. This dropsical condition may even involve the arms and the hands; fluid may be found in the pleural cavity and in the pericardial cavity. The dyspnea is severe; walking is almost impossible; the breathing is forced respiration; the patient is unable to lie down, and is compelled to sit up most of the time or in a semi-recumbent position. The cough is almost constant

with quantities of expectoration. The kidneys do but little work, and often but a few ounces of urine are passed in the twenty-four hours. Digestion is impaired; the general condition of the patient is bad; uremia often results, and pulmonary congestion becomes so severe that the heart is not able to work — all its fibers stretch, the mitral leak is increased, the heart muscle gives way and death results. This is failure of compensation with dilation.

Fortunately for these patients, complete failure of compensation does not always take place, or the threatened failure is overcome by proper treatment. In many of these cases, the right ventricle will begin to dilate, the fibrous rings to stretch, and the tricuspid orifice to enlarge, and a relative tricuspid regurgitation occurs. The jugulars pulsate; the liver enlarges and pulsates; the feet, legs and abdomen become dropsical; the kidneys are doing but little work, but the heart muscle still retains its integrity and, while stretched or dilated from overwork, it is not to any great extent diseased, and, under proper treatment, by degrees the pulse becomes fuller and stronger and more regular, and its tension increased. The kidneys begin to take up their work again; the amount of urine becomes markedly increased; the dropsy in part disappears; the venous congestion of the liver is relieved; the lungs clear up; the cough is much less; the dyspnea is not so severe; the nails and lips become a normal color, and the patient is able to go to bed and sleep. For a time he may wake up with a sense of suffocation and be compelled to sit up to, as he says, "get his breath," but by degrees these symptoms become less, and recovery more or less complete occurs, depending upon how many of

the fibrous rings that go to make the tricuspid opening contract to their normal size. This is a ruptured compensation in which recovery more or less complete takes place.

Diagnosis of Ruptured or Failure of Compensation in Mitral Regurgitation

First. The pulse becomes irregular in volume and rhythm, and of but little tension.

Second. The apex impulse as seen and felt is irregular and not distinct, and is often undulating or waving in character.

Third. The mitral systolic murmur which, during the period of compensation, was soft or blowing or musical in character, never harsh, is much decreased in force, and, while still audible, is much less distinct and sometimes lost. The pulmonic second sound has lost much or all of its snap, and, while heard distinctly, it has none of the snap that characterized it during the period of compensation. [NOTE.—The reason that the mitral murmur loses its pitch and is often lost when compensation fails is the left ventricle is not receiving sufficient blood and its walls are more or less dilated and their force decreased, so that the blood is not forced through the mitral opening with as much force, and as a result the pitch of the murmur is decreased. In other words, a loud murmur heard anywhere in the cardiac region means a good heart muscle; when a murmur that has been heard distinctly becomes less distinct, it means a loss of tone in the heart muscle, and when the pitch returns it indicates a return to the normal strength of the heart muscle.]

Fourth. When we listen at the ensiform cartilage, or between that and the apex, a soft, blowing murmur, systolic in time, that is transmitted somewhat to the right will be heard. This murmur may be transmitted to the left, but it will be lost before the apex is reached. This is a relative tricuspid murmur, the result of the stretching of the fibrous rings of the right ventricle.

Fifth. The jugulars are seen to pulsate, and the liver can be felt to pulsate, and the anasarca is more or less pronounced. Occasionally, from sudden strain, rupture or failure of compensation in mitral, regurgitation occurs very suddenly, and death results before little or no dropsy has shown itself. The lips, nails, fingers, hands, face and often the head are blue; the dyspnea is intense, the heart's action rapid and exceedingly irregular, the impulse beat scarcely or not at all discernible, the heart sounds very indistinct or lost, and death soon results.

Management and Treatment of Ruptured Mitral Compensation

Before we take up the treatment of ruptured mitral compensation, let us see what the conditions are that we have to deal with.

First. A hypertrophied right ventricle that has dilated.

Second. A poorly acting left ventricle from lack of blood.

Third. A venus stasis.

Fourth. A low arterial pressure.

Fifth. A passive congestion of and poorly acting kidneys.

Sixth. More or less dropsy.

How can these conditions be met?

First. The dilated right ventricle must if possible be stimulated so as to contract to its normal dimensions.

Second. More blood must be forced into the left ventricle and its walls made to act with more force upon the blood within it.

Third. The passive congestion of the liver and the bowels must be relieved.

Fourth. The walls of the arteries must be contracted so as to raise arterial tension.

Fifth. The kidneys must be made to act with increased activity.

Sixth. The dropsy must be removed.

How can these things be brought about? By referring to the chapter on heart remedies, we find that the drugs that stand at the head of the list in their action upon the right ventricle are, first, digitalis and its class. To a case of ruptured mitral compensation of

moderate severity, one to two drams of the infusion of digitalis, made from the fresh English leaves, may be given every two or three hours, until the urine has markedly increased in amount and quality, the force of the heart's action has become very much improved, the impulse more distinct and regular, and the breathing has improved; then lengthen the interval between the doses, giving enough to make the kidneys act freely. The digitalis acts upon the walls of the heart, increasing the force of the heart's action and lengthening the time of contraction, and it also contracts the muscular coats of the arteries and thus raises arterial tension in both the systemic and the pulmonic circulations.

The passive congestion of the liver and bowels can be relieved by some remedy that will either act directly upon the liver or indirectly upon it. The best is some form of mercury in the form of the mild chloride, the calomel to be given from one to two grains in broken doses, one-tenth to one-quarter of a grain at a dose every hour till one or two grains have been given, to be followed by some saline, as a seidlitz powder, rubinat water or some similar saline. Should the digitalis disagree with the stomach, caffeine in from one-half grain to three grains of the alkaloid may be given every two to four hours, and, should the patient be a very nervous patient, the valeriate of caffeine should be the form of the drug used. Caffeine has a direct action upon the heart muscle if the alkaloid caffeine be used, or the benzoate of caffeine be used. The alkaloid of caffeine may be given in from $\frac{1}{2}$ to $2\frac{1}{2}$ grains. The benzoate of caffeine had better be made fresh, and can be prepared by adding two drams of the benzoate of soda and

the same amount of caffeine alkaloid to three ounces of water; then a dram of the mixture will contain about five grains of the fresh benzoate of caffeine. Merck's German pure digitaline in from 1-20 of a grain to $\frac{1}{8}$ of a grain every two to four hours may be a better drug and give better results than either of the ones named. Strophanthus, cactus or nux vomica may be used, but the digitalis or the caffeine alkaloid will, as a rule, be found to be the better drugs.

With the drug treatment, special attention must be given to the diet, which should be easily digested foods. The best is skim milk, skim milk with water and a small amount of bread, rye bread being the better, some meat in the form of rare steaks or chops, or some good broth.

But the most important element in the treatment is rest. The rest should be as nearly as possible absolute, forbidding the patient to make any exertion whatever, placing him either in a Morris chair or in a bed with a suitable head-rest, so that a half-reclining position may be maintained.

Some of the alcoholic stimulants may be used and be of service — whisky or, better, Tokay wine, one to two ounces, with a little water added, every two to four hours.

In the greater number of the cases of ruptured mitral compensation of moderate severity, this treatment will be all that will be required to bring about recovery of the dilated heart, restore arterial tension, remove venous congestion, pick up the exudate in the form of the dropsy, and restore the patient to his former health. When the ruptured compensation is more severe, so that the pulse is very irregular, small

and of low tension, the dyspnea very bad, the patient not being able to lie down at all and breathe; the cough almost constant; the heart's action very irregular with a wavy impulse; dropsy very bad; the extremities, the back, the arms, the hands the nails, the fingers, the lips and face are blue, even purple — under these circumstances the digitalis should be pushed, giving from two to four drams of the infusion every two to three hours; or 1-10 to $\frac{1}{8}$ of the digitaline every two to three hours. Often with the digitalis it is best to give from one to three grains of caffeine alkaloid every two to four hours, and in addition some strychnia sulphate, from 1-60 to 1-40 every two to four hours, either by mouth or, if the case be urgent, to give it hypodermically. Should the case not improve within twenty-four hours, as shown by some increase in the amount of urine and decrease in the cough, and the dyspnea, the doses of all these drugs are to be increased; the strychnia should be given in from 1-30 to 1-25 as the case may require, every two to three or four hours, until the force of the heart's action is increased. The semi-recumbent position must be maintained and absolute rest enjoined. The diet should be of the plainest — better to be the skim milk, wine, kumyss and light broth.

If, after the heart remedies have been given for twenty-four to thirty-six hours, and attention has been given to the liver and the bowels, and the kidneys are doing some little better work, the dropsy remains severe or is increasing, other measures looking toward the removal of the dropsical fluid must be considered. Many new remedies are upon the market for this purpose, some good, and many not good, some having a

bad effect upon the heart's muscle, others not so bad an effect upon the muscle of the heart. We prefer to use the calomel treatment and can vouch for its efficacy, but each detail in its use must be followed out.

First. The digitalis must be given until the arterial tension has been raised as manifested by some positive increase in the urine. The calomel should never be given before this has been accomplished.

Second. The amount of calomel given in twenty-four hours should be nine grains, to be given in doses of three grains, three doses, one at 5 P.M., one at 7 P.M. and one at 9 P.M. Each dose of calomel is to be followed in thirty minutes by from ten to fifteen drops of the deodorized tincture of opium. The opium is to prevent the action of the calomel upon the bowels and liver and to obtain its diuretic effect alone. The three doses of calomel are to be given the second day in the same way, only it may not be necessary to give as much of the opium. On the morning of the third day a saline is to be given to carry off the excess of the drug and to clear the bowels. This is very important. Less amounts of calomel may be used, but we have never seen any harm result from its use when given in the manner described, and we have often seen less amounts of the drug given and the desired effect not obtained, therefore we advise the full amount to be given, followed by the saline. If the arterial tension has been raised as directed, and the calomel given in the manner described, the results will be very satisfactory, and in all probability this will be all the treatment that will be required to relieve the dropsy, as the kidneys will, if there be any work in them, relieve the system of a large amount of urine and very rapidly reduce the

bloating, the amount of urine often running as high as ninety to one hundred ounces in the twenty-four hours. During the time that the kidneys are acting, the digitalis must be kept up; often the dose must be increased, and once in a while it will be necessary to give some strychnia during this time, to guard against collapse. By the use of some of the more volatile heart remedies, as the aromatic spirits of ammonia in from ten to fifteen drop doses as often as every half-hour or hour, the heart's action can be very materially assisted. As a rule, after the kidneys have been once started by the calomel, they will continue under the influence of the digitalis to act very freely, and rid the patient of the dropsy. However, should they again slow up in their action, the calomel may be repeated, or small doses of diuretine, five to ten grains, may be given every two to four hours until the dropsy has entirely disappeared.

If diuretine is used, its effects upon the heart are to be closely watched, as in some cases it tends to markedly depress the heart's action, and will have to be stopped and some of the volatile stimulants given. Occasionally one will see a case where the pressure from the fluid in the tissue will be so great that it will not be possible to raise arterial tension until some of the fluid has been drawn off. This can be done oftentimes by making a small opening in the lower part of the leg with a knife, and allowing the water to drain off in that way. If necessary, the abdomen may be tapped and quite a quantity of water drawn off, but this is to be avoided if possible. Relieving the pressure in the manner just described is often enough to start the kidneys under the influence of the digitalis alone and

frequently is all that will be necessary to relieve the dropsy.

A heart that has dilated in this manner is more apt to do so again than one that has not so dilated, so that the future care of such a case is very important. The patient should be warned to guard against anything that will put a strain upon the heart or overtax it in any way, and upon the first symptoms to begin treatment at once. The treatment of sudden dilation of the heart, or sudden ruptured mitral compensation, is in the main what has been laid down, except that digitalis is not to be depended upon for rapid action. Strychnia sulphate in from 1-30 to 1-25 of a grain hypodermically as often as every hour, for several doses; caffeine alkaloid, two or three grains hypodermically every two or three hours; the aromatic spirits of ammonia in from ten to fifteen drop doses — not larger doses — every fifteen to thirty minutes. Artificial heat about the patient, and once in a while, when the right heart becomes so enlarged that its walls can not work to any advantage, bleeding will do much good. From ten to sixteen ounces of blood should be taken from the arm, thus relieving the pressure upon the right ventricle.

MITRAL STENOSIS

ETIOLOGY.—The most common cause, if not the only cause, of mitral stenosis is endocarditis or valvulitis; probably more often it is valvulitis. As a result of this inflammation, there is an exudate upon the endocardial covering of the mitral valve flaps. This exudate is often of a fibrous nature and the valve flaps become adherent, and, as the fibrous bands formed from the exudate contract, the action of the valves is greatly interfered with. By degrees the edges of the valve flaps become drawn closer and closer together, so that the mitral orifice becomes a buttonhole, or often a mere slit that the blade of a penknife can scarcely enter. These changes all bring about one result, namely, an obstruction to the blood as it passes from the left auricle to the left ventricle—a mitral obstruction or a mitral stenosis. Mitral stenosis is more common in women than in men.

CHANGES PRODUCED IN THE HEART MUSCLE, PULMONIC AND SYSTEMIC CIRCULATION BY MITRAL STENOSIS.—It will be remembered that during the period of pause of the heart the left auriculo-ventricular opening is open and that the blood is passing from the left auricle into the left ventricle, and that as soon as the ventricles are filled the auricles are filled, and then distended, then contract, and the ventricles become distended. It will also be remembered that the muscular fibers that compose the ventricles—the “figure-of-eight” fibers, surround each ventricle and that when one ventricle contracts both must contract at the same time, but when one ventricle is diseased it is possible for one ventricle to contract an instant before the other ventri-

cle contracts, but the contraction of the one ventricle is followed so closely by the contraction of the other ventricle as to leave no appreciable fraction of time between the two contractions. With this review, let us see what changes are produced in the heart muscle by mitral stenosis.

First. The blood is obstructed in its passage from the left auricle to the left ventricle during the pause of the heart, but no such obstruction exists at the right side of the heart, at the tricuspid orifice. The right ventricle will, therefore, fill before the left ventricle, and the right auricle will contract before the left auricle, and the right ventricle will be distended before the left ventricle is dilated and will, therefore, contract an instant before the left ventricle. As a result of this action, and an endeavor on the part of the left ventricle to keep pace with the right ventricle, the left ventricle will be imperfectly filled with blood and a smaller amount of blood than normal is thrown into the aorta when the left ventricle contracts. This produces a small pulse. In mitral stenosis, the left auricle is receiving a normal amount of blood from the pulmonary veins, but, owing to the stenosed condition of the mitral orifice, the left auricle, at its contraction, is unable to force a normal amount of blood into the left ventricle; the left auricle becomes overfilled with blood, and dilates and, to some extent, hypertrophies as much as its walls will permit; but no effort of the left auricle is able to overcome the obstruction at the mitral orifice, or force a normal amount of blood into the left ventricle. The result of all this is that the blood becomes dammed back into the pulmonary veins, the pulmonary capillaries and the pulmonary artery to the

right ventricle. The right ventricle must then enlarge to receive this dammed back blood, and its walls hypertrophy to force this extra amount of blood out into the pulmonary artery so as to raise the blood pressure in the pulmonary circulation, and to assist the left auricle in the work of filling the left ventricle, working against the stenosed mitral orifice.

Thus far the changes produced by stenosis of the mitral valve are the same as those produced by leakage of that valve, namely, in mitral regurgitation the left auricle is dilated by the blood that has been returned to it by the contraction of the left ventricle. In mitral stenosis, the left auricle is dilated by the blood that it has not been able to force into the left ventricle. The increased blood pressure in the pulmonary circulation is in both cases due to the blood that has been dammed back into it from the overfilled auricle. The enlargement and hypertrophy of the right ventricle is in both cases due to an effort on the part of the right ventricle to hold and to force out the extra amount of blood and to assist the left auricle in its work of filling the left ventricle. In mitral regurgitation the left ventricle is enlarged, and to an extent hypertrophied, in order to receive and to force out the extra amount of blood forced into it by the right ventricle, acting through the left auricle. But in mitral stenosis the left auricle, assisted by the hypertrophied right ventricle, may be able, with a slight stenosis at the mitral valve, to force a normal or nearly a normal amount of blood into the left ventricle, but never to overfill it, and in most cases the left auricle, assisted by the enlarged right ventricle, is not able to force a normal amount of blood through the stenosed

mitral opening into the left ventricle. As a result of these changes, the left ventricle is never enlarged in mitral stenosis, but on the contrary its walls are often contracted in order to be able to contract down upon the small amount of blood that it contains, and empty its contents into the aorta.

The physical findings in mitral stenosis are: In compensated, or fairly well compensated mitral regurgitation, the left ventricle will receive a normal amount of blood, because the left auricle is assisted by the right ventricle, and, as we know, the pulse is full or of moderate tension and fairly regular or is regular. But in mitral stenosis the left ventricle is not receiving a normal amount of blood, because the left auricle, assisted by the hypertrophied right ventricle, can not entirely overcome the obstruction at the mitral valve. In other words, compensation in mitral stenosis is never complete. The pulse in stenosis is small, and as the muscular coats of the aorta and of its branches are compelled to contract down hard upon the small amount of blood that they contain in order to maintain arterial tension, the pulse is of high tension. As the left ventricle frequently does not contain blood enough to distend the aorta when the ventricle contracts, the pulse is very irregular, often so much so as to make one wonder why a pulse at all, as a patient with such an irregularity of pulse will be in fairly good health.

In mitral regurgitation, we found that from the slight amount of hypertrophy of the left ventricle and the greater amount of the right ventricle, the apex impulse was in the fifth interspace, or under the sixth rib, more or less to the left of the normal line — depending upon the amount of enlargement of the right ventri-

cle. In mitral stenosis, there being no enlargement of the left ventricle, the apex impulse will never be lower than the fifth space, and as much to the left of the normal line as the hypertrophy of the right ventricle will push it. [NOTE.—The apex impulse is made by the striking of the apex of the right ventricle against the chest wall, and the true apex of the heart is from one to one and a half inches further down and to the left. In estimating the location of the apex, the impulse is first taken, then one to one and one-half inches are added.] The impulse in mitral regurgitation is somewhat deliberate, fairly regular and forcible. In mitral stenosis, the impulse, owing to the irregular and often insufficient amount of blood in the left ventricle and the obstruction to the action of the enlarged right ventricle by the stenosed mitral orifice, is short, distinct and irregular, often very irregular, and is a distinct tap, tap, tap — not heaving, not deliberate, not forcible, but a distinct tap, tap.

In mitral regurgitation, a systolic murmur is heard at the apex that is transmitted to the left and heard at the axillary and in the back, and sometimes to the right of the apex toward the ensiform, but it is systolic in time. In mitral stenosis, if a murmur is heard — and it is only heard in a portion of the cases — it is a high-pitched murmur and will precede the systole of the ventricle, or, in other words, it is a presystolic murmur running up to and ending with the contraction of the ventricle or the first sound of the heart. This is the most difficult murmur of the heart to diagnose, but by timing the murmur with the pulsation in the carotids and the apex beat, one will hear the murmur followed immediately by the impulse beat and the distention of the caro-

tids. If the hand be placed over the apex impulse, quite often a purr like the purr of a cat will be felt. This is called a thrill, and, as it occurs before the systole of the ventricle, it is called a presystolic thrill.

In mitral regurgitation, we hear at the apex the systolic murmur, immediately followed by the aortic second at the apex. In mitral stenosis, the fact that the left ventricle has a smaller amount of blood than normal to force into the aorta and to distend the coats of the aorta, and that the recoil of the blood from the contraction of the muscular coats of the aorta is below normal, the aortic sound is not heard at the apex or heard only at times, or very imperfectly heard. In mitral stenosis, the right ventricle is very much enlarged, and the left ventricle is not. The mitral orifice is obstructed, but the tricuspid orifice is not. The right ventricle will fill before the left ventricle, and the right will be ready to contract before the left, and in fact it does often contract before the left. This produces two sounds at the base of the heart; the pulmonic second is heard, and immediately following it the aortic second or what is called a reduplication at the base. This may also be heard over the apex. The diagnosis of mitral stenosis will be made upon:

First. The small, irregular pulse.

Second. Apex to left of normal.

Third. A presystolic thrill felt at the apex.

Fourth. A presystolic murmur at the apex not transmitted to the left but transmitted somewhat to the right toward the ensiform.

Fifth. The imperfect or absent aortic second at the apex.

Sixth. The reduplication at the base.

Seventh. The accentuated pulmonic second at the second right interspace, showing hypertrophy of the right ventricle. The clinical symptoms of mitral stenosis are for the most part those of mitral regurgitation. The cough may be more troublesome, the expectoration more abundant and often more bloody, the dyspnea more persistent upon slight exertion, the ability to lie down to sleep less; the fingers, hands, arms, feet and often the legs are blue and cold; the lips are blue; the face is pinched, and digestion is much impaired. When the fibrous rings of the tricuspid orifice begin to stretch and a relative tricuspid leak shows itself, the symptoms do not differ from those found in mitral regurgitation under the same circumstances, except that in mitral stenosis the dyspnea and cough are more troublesome and the general health more impaired.

The general management of a case of mitral stenosis is the same as that of mitral regurgitation, but people suffering from mitral stenosis can not endure as much as those having mitral regurgitation, and are compelled to take life more deliberately, and often for years are not able to lie down at night except when supported with two or three pillows. The medical treatment of mitral stenosis differs from that of mitral regurgitation. In a fairly well compensated case of mitral stenosis little or no treatment is required except to look after the digestion, the liver, the bowels and general health. Ars. Iod. 2X. Every two to four hours Ars. Alb. 3X, the same way. Stry. Phos. 2X, Stry. Ars. 2X, every two to six hours, together with good food.

Mitral regurgitation may last for years and be fairly well compensated for, but mitral stenosis is a progressive condition and often results in almost complete

occlusion of the mitral orifice. This will give a history of gradual but certain advancement with slow but sure increase of all the symptoms.

In mitral regurgitation, digitalis or its kindred drugs give best results, but these drugs are of doubtful utility in mitral stenosis. One-half to one dram may be given every two to four hours of the infusion of digitalis, made from the fresh English leaves, watching its effect on the heart and pulse very closely. If the pulse becomes more regular, the apex beat more deliberate, the aortic second present or more distinct at the apex, the drug may be continued with care, but if the pulse becomes more irregular, the apex impulse more tappy, the dyspnea more pronounced, the drug is to be stopped. The reason of this is that the digitalis slows the action of the heart and increases its force and power, compelling the right ventricle to work against an obstacle that it can not overcome, the stenosed mitral orifice, thus tending to produce dilation of the right ventricle. Digitalis contracts the muscular coats of the arteries, increasing arterial tension; in mitral stenosis, the arterial tension is already high, and the drug does harm. Digitalis should never be given in a case of mitral stenosis until the liver has been emptied by some remedy, as calomel followed by a saline. When compensation begins to fail, caffeine alkaloid in from $\frac{1}{2}$ to 2 grain doses every two hours will generally give good results. Stry. Sulph. in from 1-60 to 1-40 every two to four hours. When compensation is ruptured suddenly, the ammonia preparations will be of service. For the sudden, severe attacks of dyspnea, either in mitral stenosis or regurgitation, codein in from 1-10 to $\frac{1}{2}$ grain, or morphia in from $\frac{1}{4}$ to $\frac{1}{3}$ of a grain will give the best

and most rapid results. The calomel treatment may be used for the dropsy of mitral stenosis if it becomes severe, care being taken to raise the arterial tension with caffeine or some other drug before the calomel is given. The great importance of rest and a skim milk diet can not be too much insisted upon. The moderate use of some mild stimulant, as Tokay wine or whisky, is also to be remembered. The use of high colon normal salt enemas will be of service in stimulating the kidneys.

Mitral stenosis and regurgitation not infrequently occur together. That is to say that the valve flaps are so altered by the disease process that they permit the blood to pass back into the auricle, and at the same time they obstruct the blood in its passage from the auricle to the ventricles. The changes in the heart's muscle are the same as in regurgitation. The diagnosis is made upon :

First. The pulse, which is very irregular but with fair tension.

Second. The impulse is in the fifth space or under the sixth rib well to the left of the normal line.

Third. The impulse beat is a mixture of a tap and a normal impulse.

Fourth. A systolic murmur transmitted to the left and occasionally a presystolic murmur at the apex, transmitted to the right.

Fifth. The accentuation of the pulmonic second at the second left interspace.

The management of such cases is the same as for mitral regurgitation, only : the guarded use of digitalis.

AORTIC REGURGITATION

Mitral regurgitation is a disease of early life, and always the result of endocarditis. Aortic regurgitation, while occasionally due to endocarditis or valvulitis, and found before forty years of age, is yet most commonly caused by arterial sclerosis, and in the greater number of cases the patient is past forty and is a laborer or has been one. Inflammations of the lining of the aorta, whether from syphilis or other septic blood states, is also a cause. Occasionally ulceration of the valve cups occurs, and perforation. At other times the edges of the cups become twisted, or vegetations occur on the cups, the cup becoming adherent to the lining of the aorta. All these changes prevent the cups from properly closing the aortic opening when the ventricle ceases to contract and the recoil of the blood comes, due to the contraction of the muscular coats of the aorta.

CHANGES IN THE HEART MUSCLE AND CIRCULATION

When the aortic valves leak, permitting the blood to pass or flow back into the left ventricle after the ventricle has ceased to contract, the left ventricle is receiving blood from two sources. At this time the ventricle is relaxing and the blood is passing into it from the left auricle through the mitral opening, and if the aortic valves are leaking, the ventricle is receiving blood from the aorta through the defective aortic valves. The result is the left ventricle must dilate to hold this extra blood, and it must also increase in size or hypertrophy in order to be able to force this extra

blood out, or in other words to empty itself into the aorta when it contracts. These are the changes that take place, frequently with very extensive enlargement of the left ventricle, this condition giving the largest heart found, the so-called "ox-heart." This enlargement of the left ventricle, in uncomplicated cases of aortic regurgitation, is the only change in the heart muscle found. This enlargement of the left ventricle pushes the heart downward, not to the left. The apex will often be found in the sixth space, under the seventh rib, in the seventh space or even lower down and but little to the left of its normal line unless as otherwise described later on. This wonderful enlargement of the left ventricle gives to the heart a most forcible and powerful apex impulse. Often the whole chest, or even the body, or the bed on which the patient lies, will throb with the powerful beating of this enlarged left ventricle. The left side of the chest is often enlarged and when the hand is placed over the heart a heaving, forcible impulse is felt. The most characteristic point about this impulse beat is its deliberate action and its regularity. While, as we know, the impulse in mitral diseased conditions is often irregular, and in mitral stenosis very irregular, in aortic regurgitation the impulse is always regular. If the impulse becomes irregular in aortic regurgitation, it is a sign that the heart muscle is degenerating.

It will be remembered that in health, when the left ventricle contracts and forces its contents into the aorta, that the muscular coats of the aorta are distended, the action of these muscular coats of the aorta, acting upon a perfectly closed aortic valve, produces the circulation in the arterial systemic circulation. When the aortic

valves are leaky, and the left ventricle has hypertrophied, and this enlarged ventricle contracts, it forces into the aorta a normal amount of blood, plus the amount leaked back into the left ventricle through the defective aortic valves. This extra amount of blood when forced by the enlarged ventricle into the aorta overdistends the coats of that vessel so that the ascending portion of the aorta and often the arch become very much enlarged, in fact of an aneurismal nature. This distension of the aorta often elongates the ascending portion, so as to permit the enlarged heart to "sag" and the apex is thrown somewhat to the left. This is to be taken into consideration in forming a judgment as to the condition of the heart muscle.

In health, after the aorta has received the blood from the left ventricle, its muscular coats contract and the recoil of the blood closes the aortic valves, and the aorta empties itself in but one direction, namely, into the arterial system, but when the aortic valves are leaking the aorta empties itself in two directions: First, into the arterial system; second, into the left ventricle through the defective aortic valves. This gives to the pulse a peculiar character. The arterial system receives from the aorta a large amount of blood that has been forced into it by the enlarged left ventricle. This large amount of blood is forced into the aorta with a great amount of force. This gives a large, full pulse; but the aorta, when the left ventricle ceases to contract, empties itself in two directions, and this causes the pulse to drop or fall away or collapse suddenly. If the arm be elevated, this will be noticed more distinctly. This is what is called the collapsing pulse, the water hammer pulse or the Corrigan pulse. From the same

reason the carotids in the neck are seen to pulsate and to collapse. If a line be drawn across the forehead with blunt instrument, the line will be seen to pulsate. If the end of a finger-nail be held down lightly, the capillaries under the nail will be observed to pulsate. If the lower lip be everted and a cover glass be pressed upon it, the capillaries will be seen to pulsate. The superficial arteries become tortuous and pulsate. When the stethoscope is placed over the second right intercostal space, as a rule a murmur is heard immediately following the contraction of the ventricle. This murmur is usually heard best at the third or fourth interspace to the *left* of the sternum or down the *left* edge of the sternum. As it follows immediately after the contraction of the ventricle, it is called a diastolic murmur, and when heard is always valuable evidence of an aortic regurgitation. This murmur is often loud and prolonged and is transmitted downward toward the apex. When the stethoscope is placed over the brachial artery the femoral, and often the radial and popliteal, a distinct shock is heard, the so-called pistol shot, due to the sudden filling of the collapsed artery by the powerful action of the left ventricle throwing a large amount of blood into the aorta. From the dilation of the arch of the aorta and the ruffling of its edges, not infrequently a systolic murmur is heard at the second right intercostal space, but this murmur is not transmitted into the vessels of the neck. It will be remembered that the coronary arteries have their openings back of two of the aortic valve cups and are filled by the recoil of the blood in the aorta. As the result of the disease of the aortic valve cups, the coronary arteries do not receive their proper amount of blood

at all times, and not infrequently bits of fibrin are whipped off the arch of the aorta and are forced into one of the coronary arteries, causing sudden death. In health, upon listening over the right carotid artery, the aortic valves can be heard to close. In aortic regurgitation, when the aortic second can not be heard, or imperfectly heard, in the carotid, it denotes a bad leak of the aortic valve.

CLINICAL SYMPTOMS OF AORTIC REGURGITATION

First. The most important and the most common clinical symptom is vertigo. It is most marked when arising from a sitting position or when rising to a sitting position from a recumbent position.

Second. Pain and numbness in the arms, especially the left arm.

Third. Distress in the region of the heart; this precordial distress is as if the heart would stop beating for an instant and as though something awful was about to happen to them. Slight dropsy of the feet sometimes occurs, but it is never very bad.

The diagnosis of aortic regurgitation is made upon :

First. The pulse full, regular, high, collapsing.

Second. The apex misplaced down, perhaps some to the left, if the arch is sagging.

Third. A regular, heaving, forcible impulse.

Fourth. A diastolic murmur often heard at the second right intercostal space, but more times heard at the third or fourth left intercostal space and transmitted toward the apex.

Compensation for aortic regurgitation depends upon the left ventricle. Should, however, the aortic leak

progress to such an extent that the fibrous rings that make up the mitral opening are stretched, a relative mitral leak will occur; or should the left ventricle, from imperfect nutrition or from imperfect filling of the coronary arteries, cease to retain its integrity and dilate, the mitral rings will be dilated in the general breakdown, and a relative mitral leak result. Whenever this relative leak occurs, compensation may be said to be broken; however, not infrequently nature provides for a relative mitral leak, so as to relieve the strain upon the left ventricle. When a relative mitral leak occurs with aortic regurgitation, the changes are the same as in mitral primary leaks, namely, dilation and some hypertrophy of the left auricle, congestion of the pulmonary circulation, cough, enlargement and hypertrophy of the right ventricle and occasionally a relative tricuspid leak, pulsating veins and general dropsy. However, these latter are rare conditions. A large percentage of the people suffering from aortic regurgitation die very suddenly, from thrombus of the coronary arteries and from unknown causes; in fact, this is the usual mode of death, and friends should be so informed.

MANAGEMENT AND TREATMENT OF AORTIC REGURGITATION

People suffering from this disease should never hurry, never overexert themselves, and never allow themselves to become excited.

More mistakes are made in the treatment of aortic regurgitation than in the treatment of any other heart disease. The treatment instead of being heart tonics should be remedies to relieve the arterial tension and

thus reduce the work of the enlarged left ventricle. Such drugs as aconite 2X, codein 1-10 of a grain, every two to six hours ; nitroglycerin 1-150 of a grain two or three times a day.

When the pulse becomes irregular, which is a bad sign in aortic regurgitation, digitalis or caffeine alkaloid 2 to 3 drams of the infusion, or $\frac{1}{2}$ grain to 1 grain of caffeine alkaloid every two to six hours, watching the effect upon the pulse and the heart. Should the heart's action become more irregular, the drugs are to be stopped. Strychnia 1-60 to 1-25 every two to four hours is a splendid remedy. It is often necessary to use digitalis in large doses in these relative leaks. When a relative leak is present, digitalis or caffeine with strychnia 1-50 to 1-25 may be imperative, but they are to be used with care, remembering the tendency that digitalis has of a cumulative effect. Attention to the digestion, liver and bowels, with a light diet, whisky and Tokay wine.

AORTIC STENOSIS

ETIOLOGY.—There are two principal causes of aortic stenosis. First, arterial sclerosis; and, second, endocarditis. The valve cups become diseased, distorted, adherent, stiffened or contracted in such a manner as to prevent the blood from leaving the ventricle in a normal amount when the left ventricle contracts.

CHANGES IN THE HEART MUSCLE AND CIRCULATION

The left ventricle is receiving a normal amount of blood from the left auricle, but the aortic opening being contracted or obstructed, the left ventricle is not able to force a normal amount of blood or all its contents into the aorta. The result is the left ventricle must enlarge in order to hold this extra amount of blood and its walls must hypertrophy in order to force out this extra amount of blood and to overcome the obstruction at the aortic opening. The amount of hypertrophy of the left ventricle will depend upon the amount of obstruction that the left ventricle has to overcome at the aortic opening. The apex beat will be misplaced downward and perhaps some to the left, but very much more down than to the left. The apex is often in the sixth or seventh interspace, and it will be remembered that the real apex will be from 1 to 1½ inches further to the left and down. As a result of the obstruction of the aortic opening, the left ventricle is compelled to work hard and to prolong its contraction in order to squeeze its contents out into the aorta through the obstructed opening. This gives a forcible prolonged impulse to the heart. The prolonged forcible

contraction of the left ventricle gives a characteristic pulse.

First. The pulse is small, due to the gradual emptying of the ventricle into the aorta.

Second. The pulse is of moderate tension because the artery is not promptly filled.

Third. As the artery is not full, the pulse is low in volume.

Fourth. The pulse is prolonged, due to the slow emptying of the ventricle into the aorta, that is, the artery filling gradually; the pulse wave will come up slowly and be prolonged, and fall away slowly because the ventricle is slow in its contraction, and as its contraction is prolonged the pulse will fall away slowly. The pulse is regular, and irregularity is a bad sign. In uncomplicated cases, no other changes occur in the heart muscle. The clinical symptoms are vertigo, insomnia and often precordial distress.

The diagnosis is made upon:

First. The pulse which is low, prolonged, etc.

Second. The apex downward.

Third. The full, powerful, deliberate, prolonged impulse.

Fourth. The absence of the aortic second at the second right intercostal.

Fifth. A thrill felt over the base of the heart, systolic in time.

Sixth. A murmur, systolic in time, always heard at the second right interspace, and transmitted into the vessels of the neck, and often heard all over the chest. In the back and over the large vessels, the murmur of the aortic stenosis being often the most distinctly heard of all heart murmurs.

Cases of aortic stenosis that are well compensated and uncomplicated require but little treatment. The patients should avoid overexercise of all kinds, excitement, or anything that will put extra strain upon the heart. Should the precordial distress become severe, some remedy to lessen arterial tension, as aconite or nitroglycerin in small doses, should be given, care being taken not to give enough of the drug to produce an effect on the heart muscle so as to weaken its walls. But uncomplicated cases of aortic stenosis are rare. As a rule, the left ventricle hypertrophies to such an extent as to stretch the mitral rings, and a relative mitral leak results; then the symptoms of mitral regurgitation are added to those of the aortic stenosis. The right ventricle hypertrophies, the apex becomes pushed to the left, but is still more down than to the left. A relative tricuspid leak may result, and dropsy be present.

The treatment of such a case will be digitalis, caffeine alkaloid, strychnia, morphia or codein to relieve the dyspnea, and otherwise the same care that a case of mitral regurgitation should receive.

TRICUSPID REGURGITATION

Primary tricuspid regurgitation is a rare affection and is always congenital, due to interuterine endocarditis. When found, it can be diagnosed:

First. By a systolic murmur heard best at the fifth left space transmitted downward and to the left, gradually decreasing in force.

Second. Systolic venous pulsations in the jugulars.

Third. Intense cyanosis.

Fourth. Dropsy more or less complete.

RELATIVE TRICUSPID REGURGITATION

Relative tricuspid leaks have already been spoken of, and are due to stretching of the fibrous rings that make the tricuspid opening, from dilation or excessive hypertrophy of the right ventricle.

TRICUSPID STENOSIS

Tricuspid stenosis is one of the rarest, if not the least frequent, of heart lesions. Its presence is indicated by a thrill over the tricuspid area, presystolic in time, and a presystolic murmur over the same locality. No special clinical symptoms are present.

PULMONARY REGURGITATION

This is a very rare condition, indicated by a diastolic murmur heard best at the second left intercostal space, not transmitted to the apex, and the second aortic is audible at the second right space. No changes

in the pulse, there is hypertrophy of the right ventricle, but not of the left, as in aortic regurgitation. No special symptoms and no special treatment.

PULMONARY STENOSIS

This is a rare congenital lesion that is more common in women than in men, and more particularly in women who have incipient or acquire pulmonary tuberculosis. The right heart is enlarged. There will be a history of cyanosis from birth. A systolic thrill is felt over the second left intercostal space and a loud systolic murmur is heard at the same place that is transmitted into the veins of the neck. No special treatment is required.

FUNCTIONAL HEART MURMURS

The murmurs that we have been considering are the organic murmurs due to:

First. Disease of the valves.

Second. To stretching of the fibrous rings, stretching the mitral or tricuspid openings.

These are the so-called organic or endocardial heart murmurs. But not every murmur heard over the heart is due to one of these causes. Murmurs are sometimes heard at the base of the heart, and some of them are transmitted into the veins of the neck or in the subclavian vein, and are almost always systolic in time. The greater number of functional murmurs are heard over the pulmonary area, the second left intercostal space. They are as a rule soft and blowing in character and are heard more distinctly at the end of inspiration, while organic murmurs are heard best at the end of expiration. These functional murmurs are apt to be associated with anemia and are not accompanied by changes in the heart muscle or the pulse. In this connection let attention be called to the great importance of locating changes in the heart's muscle and of the danger of making a diagnosis upon the murmur alone; in fact, the murmur should be confirmatory evidence of the changes found in the heart's muscle.

DIFFERENTIAL DIAGNOSIS

Mitral regurgitation and mitral stenosis are to be differentiated from each other. The changes in the heart's muscle are practically the same, namely, enlargement of the right ventricle, as shown by the apex misplacement to the left, but in regurgitation the apex is somewhat downward as well as to the left, but much more to the left than downward, while in stenosis the apex is to the left and but little if any down. In regurgitation, the pulse is fairly full, fairly regular or very regular, and of low or moderate tension. In stenosis, the pulse is small, of high tension and very irregular. The heart impulse in regurgitation is fairly strong, regular and has some force. In stenosis, the cardiac impulse is rapid, "tappy," irregular and with but little force. The murmur in regurgitation is systolic in time, is soft and blowing or musical in character, beginning with the first sound of the heart, and is most distinct at its beginning, and loses force and pitch as it runs. It is transmitted to the left and heard in the axillary and under the spine of the scapula. The murmur of stenosis is presystolic in time. It begins on a low pitch or as a roll and gradually increases in force, running up to and ending with the first sound of the heart, and is transmitted to the right. In mitral regurgitation, the aortic second is always heard at the apex. In mitral stenosis, the aortic second is lost at the apex, only occasionally heard or imperfectly heard. In regurgitation, the arterial circulation is good. In stenosis, the hands, arms and feet are cold and often blue.

Aortic and mitral regurgitation are to be differen-

tiated from each other. In aortic regurgitation, it is the left ventricle that is enlarged, the apex is down, not much to the left. If to the left, is due to the "sagging" of the arch of the aorta, and the apex is much more down than to the left, while in mitral regurgitation the apex is much more to the left than down. The cardiac impulse, in aortic regurgitation, is deliberate, forcible and distinct, while in mitral regurgitation it is much less forcible and more rapid. In aortic regurgitation, the pulse is full, high and collapsing. In mitral regurgitation, the pulse is never collapsing. In aortic regurgitation, the murmur is diastolic in time and heard best at the third or fourth space to the left of the sternum, transmitted downward toward the apex, but is never heard in the axillary or under the angle of the scapula. In some few cases of aortic regurgitation, the murmur will be heard at the apex, or just to the right of the apex as a presystolic murmur, and is called the Austin Flint murmur, after Austin Flint, who was the first to call attention to this condition. The murmur of mitral regurgitation is always systolic in time and is transmitted to the left, and while often heard to the right of the apex is also heard to the left and behind as well as in the axillary. It is to be remembered that a systolic murmur is often heard with aortic regurgitation at the base, this murmur being due to the roughening of the arch of the aorta.

Mitral regurgitation and tricuspid regurgitation are sometimes to be differentiated between.

The changes in the heart's muscle are the same. In mitral regurgitation alone the murmur is transmitted to the left, sometimes to the right; if so, the pitch of the murmur decreases as the distance from the apex

increases, while in a tricuspid murmur the pitch of the murmur decreases as the distance to the apex lessens. Dropsy does not occur with a mitral murmur until the tricuspid valve leaks. It is not always possible to hear a tricuspid murmur or to differentiate it from a mitral murmur, but if in a given case it is known that the mitral valve has been leaking and the right ventricle has been enlarged and dropsy shows itself, we are sure that the tricuspid valve is leaking although we may not be able to hear a murmur.

HYPERTROPHY AND DILATATION OF THE HEART

Hypertrophy of the left ventricle will result from any increased resistance in the systemic circulation as :

First. Aortic regurgitation.

Second. Aortic stenosis.

Third. Disease of the kidneys.

Fourth. Vaso-motor conditions.

The physical findings of hypertrophy of the left ventricle are :

First. The enlargement of the left ventricle as shown by the downward misplacement of the apex.

Second. When the aortic valves are not diseased the aortic second at the second right interspace will be distinctly accentuated.

Third. A forcible impulse beat.

The treatment is confined to the diseased condition causing the enlargement.

Hypertrophy of the right ventricle will result from any increased resistance in the pulmonary circulation as :

First. Disease of the mitral valve or its orifice.

Second. Disease of the lungs, as chronic phthisis.

. The diagnosis will be made :

First. Upon the misplaced apex to the left, somewhat downward but much more to the left.

Second. Accentuated second pulmonic at the second left interspace.

Third. A strong impulse.

Dilation of the heart may be acute or chronic. Acute as the result of some sudden strain upon the heart, as a boat race, mountain climbing, etc. The heart's action will be feeble and rapid, although the impulse may be distinct, but it will be wavy in character and markedly irregular, and the heart's sounds are very indistinct or lost, the pulse weak and irregular, the nails blue, the face cyanotic, and dyspnea great. The treatment has been given in speaking of failure of compensation in mitral regurgitation.

ENDOCARDITIS

Endocarditis may be acute or chronic, but it will always be the result of some infective blood state. The more common being acute articular rheumatism, measles, diphtheria, pneumonia or septicemia. The endocardial covering of the valve flaps or cups may be alone affected, a valvulitis, or the endocardial covering of the valve flaps and the papillary muscles or the chordæ tendinæ may be involved, and rarely the endocardial lining of a portion of the ventricle or even in some rare cases the endocardial lining of the entire heart.

Interuterine endocarditis affects the right side of the heart only. Post-uterine endocarditis usually, if not always, affects the left side of the heart. Acute endocarditis may be accompanied by some rise in temperature or increase in temperature over that the patient has been having. The pulse may be irregular; they may have some dyspnea, a slight cough and a soft, blowing murmur at the apex and in rare cases at the aortic valve.

The treatment is rest in bed and, in addition to the remedies that are indicated for the existing conditions, such remedies as will tend to reduce the temperature. Some heart tonics may be called for and, if so, digitalis in small doses, say from a dram to two drams three or four times in the twenty-four hours, or one grain of caffeine alkaloid two or three times a day. But the most important element in the treatment is rest in bed until compensation has taken place, as manifest by the hypertrophy of the right or left ventricle, depending upon the valve involved. Chronic endocarditis, which

is almost always a valvulitis, has no distinctive symptoms except the gradual deformity of the mitral and occasionally of the aortic valve. The deformity will be a gradual leakage or stenosis of the mitral or the aortic valves, and the enlargement of the heart will go hand in hand with the deformity of the valves.

The treatment of chronic endocarditis is the treatment of the valvular defects that result from it.

PERICARDITIS

Pericarditis may be serous, sero-fibrinous or purulent.

ETIOLOGY.—Pericarditis is the result of some toxic blood state, as acute articular rheumatism, measles, diphtheria, septicemia, Bright's disease, extension of inflammation from the lungs, the pleura, mediastinal glands, ribs or sternum.

PATHOLOGY.—In serous pericarditis, the lining membrane of the pericardium and that covering the heart become highly congested and inflamed; the normal secretion is checked, so that when the heart acts bringing its surface in contact with the lining of the pericardial sac friction results. In from a few hours to twenty-four or thirty-six hours, the dryness gives place to exudate, and an excess of secretion is found, putting a stop to the friction. The amount of fluid will vary from a dram or two to several ounces. Once in a while no fluid is found — the so-called dry pericarditis.

In the sero-fibrinous form, the congestion of the lining of the pericardium is the same, but the exudate is fibrinous in character. The heart and the lining of the pericardium are covered with bits of fibrin, giving the heart a peculiar appearance — the so-called shaggy heart. In this form of the disease there is never much fluid. The deposits of fibrin often form bands of adhesions between the covering of the heart and the lining of the pericardial sac, and the surface of the heart becomes adhered to the lining of the pericardium, "adhered pericarditis." Again, the inflammation of the pericardium extends through the sac and involves the pleura adjacent to it, and adhesions may occur,

pluro pericardial adhesions. These adhesions may distort the shape of the heart, or interfere with its action or cause misplacements.

In the purulent form of pericarditis, the exudate is pus containing various forms of bacteria and varying in amount from a dram to several ounces. Very often the inflammation of the pericardial sac extends into the heart muscle, and a myocarditis exists with the pericarditis.

SYMPTOMS AND CLINICAL HISTORY

In pericarditis, the temperature is somewhat increased, from one to two or even three degrees. If the disease occur as a complication of some other pathological state, the temperature will be increased and the pulse will be higher; some slight, dry, hacking cough will be present, some dyspnea, and inability to lie down with comfort. Precordial distress will be present and in some cases this distress is severe.

The diagnosis of acute pericarditis is dependent upon one symptom, namely, a to-and-fro friction sound heard best at the fourth or fifth left interspace. This friction sound is harsh, rough or grating in character, and is usually a to-and-fro sound occurring with the contraction of the ventricles and the diastole of the heart. Sometimes it is only heard with the contraction of the ventricles. Other times it is triple, occurring with the contraction of the ventricles, with the diastole of the heart and with the contraction of the auricles. This sound, when listened to, seems to be very near the ear, and is much increased by pressure with the bell of the scope. It is not influenced by respiration and, as a rule, is much increased by leaning forward

or sitting up. This sound may not be present at all or it may last but a few hours, or it may last, if the case be a dry pericarditis, for days or even longer. When this friction sound is found, it is positive evidence of pericarditis. Sometimes the exudate will occur without any friction sound having preceded it. When fluid accumulates in the pericardial sac the heart sounds become muffled or indistinct, the impulse weak or not at all perceptible, while the pulse remains good — this phenomenon being more noticeable when the patient is sitting up. Occasionally the pericardial sac will contain a very large amount of pus (this is especially true in the case of children), the precordia will be distended, the interspaces bulging and dulness very marked. If the exudate be pus, it may be suspected by the general signs of pus and signs of pericardial fluid.

TREATMENT OF PERICARDITIS

In addition to the treatment of the general condition that gives rise to the pericarditis, some remedies to relieve the temperature, as Aconite, Bell. or Bryonia 1X. To relieve the friction pain and to control the inflammation, the ice bag gives the best results; pounded ice with salt in the hot water bag or in an ice bag. Bryonia 1X; five to ten drops every hour is an excellent remedy. After the friction sounds have disappeared, dry heat will be the best. It is often necessary to give some remedy to control the pain, and Murc. Sol. 2X, and Morphia Sulp. 2X, one to two grains of the powder every hour or two will do good. Codein, 1-10 of a grain, every hour or oftener if necessary. After the acute symptoms have subsided, some of the

heart tonics, as digitalis or caffeine alkaloid, in small doses, Ars. Iod. 2X, may be of service. Should the amount of fluid be great, aspiration must be performed. The skin is prepared and an incision is made with a knife through the skin and underlying tissue down to the interspace, 1 to 1½ inches to the left of the sternum; the needle is pushed in straight close to the upper edge of the rib until it enters the pericardial sac, then the hand is pressed down to the abdomen and the needle pushed upward. No harm can come from pricking the heart if but little force is used, should this accident happen.

When pus is present, the same procedure is followed, only a trocar in place of an aspirator is used; the pus is drawn off, and drainage inserted. The pericardial sac is best washed out by placing the patient in a tub of water above the drainage, and allow the heart to wash out the sac by its action. Nothing special can be done for the adhesions. Should myocarditis and dilation of one or more of the chambers of the heart take place, great care must be taken until compensation has become complete.

MYOCARDITIS

Myocarditis is an inflammation of the heart muscle.

ETIOLOGY.—Extensions of endocardial or pericardial inflammations. Infection from toxic blood states, granular infiltration of the heart muscle as found in typhoid fever, the so-called cloudy swelling, fatty infiltration, fatty degeneration.

Myocarditis may be acute or chronic. In acute myocarditis the heart action becomes very rapid and irregular, the impulse feeble and tappy in character, more like a push, and the apex may become misplaced. The first sound as heard at the apex becomes short and weak until the first and second sounds at the apex become of the same duration, but the second sound will have the most snap. In other words, from the degenerative changes of the heart muscle, the first sound loses its muscular element, while the second sound retains most of its snap. The pulse is weak and irregular, the heart's impulse rapid and irregular, a "tick-tack" heart and pulse. With this there will often be a systolic murmur at the apex.

The symptoms of myocarditis are great exhaustion on even the slightest exertion, added to the previous existing conditions and symptoms, inability to lie down, cough, dyspnea, synosis. The treatment is absolute rest and attention to the general condition. Heart remedies are to be used with care, for fear of producing dilation or cardiac aneurism. Acute myocarditis is a very rapid and fatal disease, and recovery, if at all, is slow. Malignant myocarditis is usually an extension of a similar condition from the endocarditis or the pericarditis and can not be correctly diagnosed.

Sclerosis of the coronary arteries gives rise to fatty or fibrous changes of the heart muscle. Chronic toxic blood states may also cause fatty degeneration. The recognition of chronic myocarditis, especially of fatty changes, is an exceedingly difficult matter and is at best but a conjecture. The history and symptoms of the case will be of importance in forming a judgment. "When a man past forty begins to have a weak heart, as manifest by a rapid, irregular pulse, with the first and second sounds at the apex about equal, or the second sound even more distinct at the apex, who has arterial sclerosis, or has had a high arterial tension or has used liquor to excess or has had syphilis and his heart has a 'gallopy' rhythm with soft, blowing murmur, we may suspect chronic myocarditis."

The treatment is rest, with such remedies as *Ars. Iod.* 2x, *Ars. Alb.* 2x; digitalis in small doses, and strychnia may be used with care.

Fatty deposits may be suspected when the patient has grown very obese; the heart sounds are very weak; the pulse is slow — 50 or 60 — but will run up to 90 or 100 upon the slightest exertion.

No treatment is of any avail except attention to the general condition. Fatty degeneration has no definite symptoms by which it can be known. Signs of chronic myocarditis, together with a tendency to deposits of fat in other organs, may lead us to suspect fatty degeneration.

TACHYCARDIA OR RAPID HEART

Rapid heart may be congenital and a pulse of 90 to 100 may be a normal pulse, but tachycardia is sudden attacks of rapid heart action, in which the action of the heart will run from 120 to 200 per minute for from a few moments to several hours. The attacks of rapid heart action may occur with organic disease of the heart, or as a pure nervous condition. The cause of these rapid actions are unknown, some nervous condition that we know nothing of. The treatment is rest, usually in the recumbent position, attention to digestion, the liver and the bowels, and any remedy that may be indicated, as *Ars. Alb.* 2x, *Aconite*, *Bell.*, *Cactus*, *Gels.*, etc.

BRADYCARDIA OR SLOW HEART.—This condition may be due to exhaustion. It often follows after fevers, as typhoid, severe exhausting labors, toxic states, autointoxication, jaundice, uremia, disturbances of digestion or hysteria. Increase in the intercranial circulation or pressure as from cerebral hemorrhage, fracture of the skull, meningitis or effusions within the cranium.

Coronary sclerosis or chronic myocarditis may give rise to permanent slow heart — forty to fifty, for years — without any other symptoms. Irregular action of the heart may be due to organic disease, adherent pericarditis, emotional conditions or excessive exertion or digestive disturbances. The prognosis will depend upon the cause of the irregularity. The treatment is the treatment of the disease condition producing it.

PALPITATION

Palpitation or an irregular heart action which is noticed by the patient may be caused by: First, nervous conditions. Second, toxins, as tobacco, coffee, tea, drugs. Third, digestive disturbances. Fourth, chronic constipation. Fifth, reflex pelvic conditions. Sixth, organic disease of the heart. As a rule, palpitation means a nervous condition, digestive disturbance, constipation or pelvic reflex.

The treatment, when it does not depend upon organic disease, is the treatment of the cause.

ANGINA PECTORIS

In the diseases of the heart that we have so far considered, pain has not been a prominent symptom. While we found that those suffering from aortic disease often have precordial distress and pain and numbness in the arms, especially the left arm, still this pain is not often severe, and at no time does it threaten life. But in angina pectoris, the pain is the most prominent symptom. The patient has been engaged in some unusual exercise, or he has been walking against a strong or a cold wind. There are no warnings of the approach of the attack, but in an instant the pain attacks him. This pain is in the chest and down the arms, and is of the most intense severity, compelling the patient to stop in the position in which he was when the pain came on. With this pain there is the most intense fear of something about to happen, of death, of unconsciousness, or of something indescribable. The face, the forehead, hands, and often the entire body, are covered with a profuse perspiration. After an attack, these patients describe the pain as if the heart was in a vise, or a ton weight was on the chest, crushing the chest. They say that the pain was so severe that another second would have killed them. At other times, the pain begins in the wrists and extends up to the arms and the chest, and often up to the neck. After the pain has ceased, they will not move for fear that the pain will return, often remaining for hours in the same position rather than take a chance of its return. Often with the passing of the pain a small amount of gas will escape from the stomach, and this fact will give rise to the belief that the stomach is the cause of the

attack. These attacks of pain do not last for longer than a few seconds, or at most for a few moments. When the attacks occur in the night without any provocation they are apt to last longer. When the attack comes on from exertion, as a rule as soon as the exertion ceases the pain is relieved. But all of the attacks of angina are not as severe as that which we have described. After these patients have a few attacks they learn to know the symptoms, and as soon as they feel the first symptom they stop, or sit perfectly still and thus cut short the paroxysm, which may end with simply a pain in the arm or some pain in the chest, but not the severe pain and the awful dread and fear. Some of these patients have attacks of angina for years, at varying intervals, then the attacks cease and they have in their place fainting spells, and finally die in one of the fainting spells. These attacks may be brought on by any little exertion, as stooping, opening a drawer or a window, or exposure to cold winds; again, indigestion or constipation may cause them. They are apt to come on in the night—the contact of the cold sheets or getting uncovered in the night or sitting up in bed suddenly may bring them on. The exciting cause of the attack is evidently a demand upon the heart for extra work. As to the cause of the attacks much speculation has been indulged in. We know some things about them, but not all. First attacks of angina never occur with mitral disease, and people have been known to have angina and afterward found to have developed a mitral lesion, and never have another attack of angina. As has been pointed out, attacks of angina may occur with aortic disease, but as a rule they are not so severe and are not accompanied

by the awful dread of something about to happen. Gout has been known to cause the disease, and disease of the coronary arteries is often found when angina has proven fatal, as well as changes in the myocardium. As to just what the state of the heart is during the attack is impossible to say. It is certain that the direct cause is a demand upon the heart for extra work that it is not able to do, and this demand upon the heart evidently brings about an unequal or irregular contraction of the heart muscle, perhaps as one has said, "like an hour-glass contraction." One cause of this extra demand upon the heart is persistent high arterial tension.

DIAGNOSIS.—There can be but little difficulty in making a diagnosis of true angina ; the extreme anguish and the pain with a cause in some sudden exertion, or a history of a previous attack will enable one to make a diagnosis of the disease. If the first attack comes on during repose and after a meal, it is in all probability a pseudo angina. Hysterical pseudo angina often occurs, and, while these women may complain of great precordial pain, they do not have the fear of something about to happen to them. The prognosis in angina is always uncertain. One attack predisposes to another and any one of them may prove fatal.

TREATMENT

First. The treatment during the attack.

Second. The treatment during the intervals of the attacks.

The treatment of the attack is first to relieve the heart by relieving the capillary tension. This can best be done by the inhalation of nitrate of amyl, chloro-

form or nitroglycerin. The amyl nitrate can be carried with them in the shape of glass capsules that can be broken in the handkerchief and inhaled when the attack comes on, without loss of time as soon as the first symptoms show themselves, or the amyl nitrate can be carried in a small vial and a few drops put upon the handkerchief and inhaled. The action of the nitroglycerin is not so rapid as that of the amyl nitrate, but its effects last much longer. Tablets containing one mīm of the one per cent solution are about the right amount, and one or two of these can be taken as soon as the first symptoms show themselves, and, if necessary, the amyl nitrate can be inhaled until the nitroglycerin has had time to take effect. When the pain and precordial distress continue in spite of the nitrates and aconite which is often indicated, morphia $\frac{1}{4}$ hypodermically, and atropine $\frac{1}{150}$, may be of service. These patients are to be warned never to use the nitrates unless they are sure that an attack is coming on, because the action of the nitrates is to relax the muscular coats of the arteries and at the same time they act in a similar way upon the heart's muscle and, if they be persisted in, they may have a bad effect upon the heart's muscle. After a severe attack, rest in bed is imperative, with a light diet and some stimulant, as the Tokay wine, $\frac{1}{2}$ to 1 ounce every two to three hours, with some light food and attention to the digestion and the bowels. The return to the usual occupation should be by degrees, first sitting up for a time one day; if this is accompanied by vertigo, rest again in bed or in a semi-recumbent position, and again sit up, then walking for a little distance, great care being exercised not to overdo. These patients soon find that they can do more one day

than they can do on another day, and soon learn to look after themselves in the amount of exercise they can take.

TREATMENT BETWEEN THE ATTACKS.—It will be remembered that the direct cause of the attack is the inability of the heart to stand the strain put upon it, and that the direct cause of this strain is an abnormal increase in the arterial tension. We must, therefore, then endeavor to find the cause of this vaso-motor spasm and do what we can to relieve it or prevent it. If the vascular contraction be due to disease of the coats of the arteries, nothing but attention to the general condition can be done. If, however, the vaso-motor spasm be due to the excessive use of narcotics, as tea, coffee, or tobacco, their use should be stopped. If mental worry or anxiety, coupled with the excessive use of one or more of these narcotics, it should be removed. Change of occupation from that of the city to the farm and its quiet, or a small town, may put a stop to the attacks. Sexual excesses often play an important part in the cause of the attacks.

HEART REMEDIES

In the following pages we shall recommend certain drugs to be used in a certain way for certain diseased conditions of the heart and, while we have no doubt but that the suggestions herein made will meet with much criticism and from some sources raise a storm of opposition. We believe that the heart is an organ, or more properly, a mechanical pump, upon which so much work is thrown and of which so much work is required, and that any defect in its mechanism that can not be remedied by nature or compensated by her efforts must be met in a mechanical way, and, as the act of propelling the blood into the systemic and pulmonic circulations is a mechanical act, we have to deal with altogether a different proposition than in the treatment of any other organ or set of organs in the body. The liver or the kidneys depend upon cell action. Their work is secretion and excretion, some mechanical, some part manufacturing. The stomach is dependent upon its glands and their activity for its juices and ferments. But with the heart all this is different. Its walls are compelled to lift so much blood pressure so high, so often, and under such a blood pressure, and to empty themselves so often, in order to be able to receive the incoming blood and thus maintain the circulation. In other words, we have a mechanical organ required by nature to do so much work in so much time and provided for by nature, that in case one or more of its parts should become imperfect, to a great extent compensate for the imperfection. Nature has also provided certain well-known remedies whose action upon the heart are known and are to be considered in the light of helps or aids to

a defective mechanical organ. As in a machine a hot-box will derive but little good from a few drops of oil, or an overworked part but little assistance from a wire wound about it, so in the consideration of heart remedies it is physiological action that is required, not dynamic action. While the dynamic action of drugs will be of service in neurotic heart conditions and is of service in acting upon blood vessels, the use of the so-called heart remedies is very much abused and often injurious. Because a valvular defect has been discovered is no reason why digitalis should be given. These remedies may be indicated or they may be very injurious. Let us then take up the consideration of a few of the most frequently used and indicated remedies and endeavor to obtain a clear understanding of their action and their sphere of usefulness.

ACONITE.—This drug is of service when, as in angina pectoris, there is great precordial distress, accompanied by intense anxiety. In aortic disease, when the pain and numbness extends down the arms and the patient is extremely anxious and fearful.

ARS. ALB.—Is indicated in heart conditions, when the characteristic indications for the drug are present, as the dry, scaly state of the skin, marked thirst, intense restlessness, inactivity of the kidneys and some dropsy of the feet and legs.

ARS. IOD.—Is an excellent heart tonic where the nutrition of the heart muscle is involved, as after fevers or exhaustive diseases of any kind. It is also often of service in mitral diseases of small extent.

AMMONIA.—Of the volatile heart remedies, ammonia is the most frequently used and the most reliable. The aromatic spirits or, if the patient be very nervous,

the valerianate, in doses of ten to fifteen drops, given in water, is a powerful heart stimulant and acts very quickly. In the use of ammonia salts, it is to be borne in mind that they are very quickly eliminated from the body by the lungs and the kidneys, so that if their continued action is required the dose should be repeated very often, even as often as every fifteen to twenty minutes. In failure of compensation from any cause, the ammonia salts are very reliable given per mouth, if the dose be repeated often.

BRYONIA.— This drug is one of the best at our command for the pain of pericarditis, but should be used in from five to ten drop doses of a good tincture, and should be given in a capsule.

BELL.— Is of service in tachycardia or other rapid or irregular heart conditions, when the pupils are dilated and the face flushed and the patient is more or less irritable.

CRATAEGUS.— Is a heart remedy that deserves much more use than it receives. It is very much like digitalis in its action when given in from ten to fifteen drop doses of the tincture. This drug does not contract the coats of the arteries to any extent, making it a useful remedy in aortic disease and in stenosed conditions of the mitral valve.

CACTUS.— Is a drug much spoken of, but as a heart tonic it is not to be compared with digitalis or its class. In pseudo angina or in some attacks of precordial pain, as often accompanies aortic disease, this drug will be of service to relieve the strain upon the heart. The indication for its use is the griping feeling as if the heart were held in a vise.

CODEIN.— This drug is often of great service when

the attacks of dyspnea are severe. It may be used in from 1-10 to $\frac{1}{2}$ grain as often as every hour or two. It is also of service in aortic disease when the left heart is threatening to dilate and the precordial distress is great. Codein given in small doses 1-10 grain every two hours will relax the arterial tension so as to relieve the strain upon the left ventricle, and often save the heart from dilating.

CAFFEINE.— This drug is much used as a heart tonic. The alkaloid is a heart tonic and has a direct action upon the heart muscle and acts very much like digitalis, only not so much upon the blood vessels. Most users of caffeine use the citrate, which probably has no action upon the heart muscle at all, but acts upon the kidneys and is a diuretic of much value. To obtain the tonic action of caffeine, the alkaloid in from $\frac{1}{2}$ grain to 2 grains at a dose is to be given from two to four hours apart. The fresh benzoate of caffeine, made by taking equal parts of the benzoate of soda and caffeine alkaloid, giving from two to three grains of the mixture at a dose and repeating it if necessary every two to three hours. Caffeine used in the manner described is a very valuable drug and one that can be used when digitalis is indicated, but the patient can not tolerate the digitalis on account of the disturbing action of that drug upon the stomach. The great mistake in the use of caffeine is the use of the citrate, and expecting action upon the heart muscle. The valerate of caffeine will often be of service as an aid when the kidneys are inactive and the patient is extremely nervous.

DIGITALIS.— Various preparations of digitalis are in use, but the most reliable is the infusion made from the fresh English leaves, which can be given in from a

dram at a dose to two or even four drams at a dose, and as often as every two or four hours. Merck's pure German digitaline to be used in doses of 1-16 to $\frac{1}{8}$ every two to four hours. The foxglove is the most universally used of all the heart remedies, and it is one of the best, if not the best, heart tonic known, and one of the most frequently abused remedies. It has its field of usefulness, and there are conditions in which it is very harmful. Digitalis stimulates the entire cardio-vascular system, producing an increase in the force and power of the heart's action. The contractions are increased in force and lengthened, and the time between the contractions is increased. It, therefore, causes the heart's action to become slower, the contraction to become more deliberate and forcible. Digitalis also acts upon the muscular coats of the arteries, contracting them and stimulating them to a more vigorous and prolonged contraction, thus raising the blood pressure in the arterial and the pulmonic circulations. Digitalis will be indicated whenever it is desired to slow the heart's action, and no contra indications are present. Whenever it is desired to raise arterial tension and no contra indications are present. In what organic heart lesions, then, will digitalis be indicated? First: In mitral regurgitation, when the right ventricle is not properly maintaining the blood pressure in the pulmonary circulation and assisting the left auricle in its work. Digitalis will slow the heart's action and increase the force and the power of the contractions and at the same time increase the blood pressure in the systemic circulation and thus increase the action of the kidneys. In mitral regurgitation, when the right ventricle has been overworked by extra strain

put upon it, and its fibrous rings are stretched and the tricuspid valve is leaking, the pulmonary congestion is increased and the pressure in the venous circulation is increased and the blood pressure in the systemic circulation is decreased, due to imperfect filling of the left ventricle and imperfect action of that chamber, and the blood serum has exudated into the lymph spaces. Digitalis is indicated, as it will increase the force and power of the heart's contraction, thus raising blood pressure in the systemic and the pulmonary circulations, slow the heart's action and increase the activity of the kidneys. When digitalis is used in mitral regurgitation, the first noticeable effect is upon the kidneys, increasing the amount of urine, and this result is to be obtained if the beneficial action of the drug is to be had. It may be necessary to increase the dose, but that should be done till the kidneys feel the effect of the drug. In mitral stenosis, digitalis must be used with great care. The right ventricle and the left auricle are working against an obstacle that they can not overcome, in the shape of the mitral obstruction. To give digitalis to such a case is running a risk of increasing the force and power of the right ventricle to such an extent as to cause it to dilate. In mitral regurgitation, the right ventricle has no obstruction ahead of it that it may not overcome. Then in mitral stenosis the tension in the arterial systemic circulation is high, and the use of digitalis is apt to increase the already high tension. If digitalis is used in mitral stenosis, it is to be used with great care, and if the pulse and heart become more irregular, its use is to be discontinued. In aortic stenosis and aortic regurgitation digitalis is never indicated unless a relative mitral leak is present. In

aortic regurgitation the use of the drug will increase the force and power of the already overacting left ventricle and at the same time contract the muscular coats of the aorta and the arterial system, and thus increase the resistance that the left ventricle has to meet and increase the aortic leak, as this depends upon the contraction of the muscular coats of the aorta. In aortic stenosis, digitalis, by increasing the force and power of the already overacting left ventricle that is working against an obstruction that it can not overcome, tends to produce dilation of the left ventricle. When a relative mitral leak is present with aortic valve disease, digitalis is indicated up to the relief of the mitral leak. Digitalis is the best remedy to relieve a relative tricuspid leak in gradual dilation of the heart. Digitalis will often disturb the digestion and, after it has been given for a time, will have to be stopped on account of its action upon digestion. From this fact it may be necessary to give some other drug for a time or substitute some other remedy altogether.

IODINE.—This drug is often indicated when a mitral systolic murmur is found with chorea, and will often give excellent service.

LYCOPodium.—This drug is often of service in irregular heart action, when the cause of the irregularity is digestive disturbances.

MORPHIA.—Is not considered as a heart remedy, but it is often of very great service in relaxing the arterial tension in angina, and is of service in the severe attacks of dyspnea from ruptured compensation. In shock, morphia is a valuable heart tonic. It quiets the nervous condition of the patient and at the same time acts to an extent upon the heart.

NUX VOMICA.— Has an action like that of strychnia only not so great. It is of use more as a general tonic. In mitral disease, when the right ventricle begins to let down, as manifest by the inability to lie down for the night, nux vomica is often a very useful drug.

NITROGLYCERIN.— Is usually classed as a heart remedy, but it is not. The drug dilates the capillaries and, to some extent, the arteries, and in this way relieves the strain upon the left heart. Its indications are labored, forcible or irregular action of the heart. In angina or the precordial distress of aortic disease, it will give relief. The dose is 1-100 to 1-50 grain every hour or two, or oftener if the case demands it, for a few doses.

STROPHANTHUS.— The action of this drug is very much like that of digitalis and its class, only it is said not to act to such an extent upon the blood vessels and does not disturb the stomach. When digitalis is not well borne, and other remedies have been tried and have not served the purpose, strophanthus may, and quite often will, give good results. The dose is from one to ten drops of the tincture.

STRYCHNIA.— Of the heart stimulants, strychnia stands at the head of the list. The forms of the drug used vary, some preferring one form of the drug and some another. Stry. Sulp., Stry. Ars., Stry. Phos., Stry. Nit. Perhaps the sulphate is the better drug to use when the stimulant effect is desired, and the other forms of the drug when it is desired to obtain more of a tonic action and at the same time to act upon the general nervous system. Strychnia acts through the nervous system, producing increased activity of the entire heart's muscle. The force and power of the

heart's action is increased. It also increases the arterial tension and has a decided action upon the venous circulation. Strychnia is the best remedy to produce rapid and efficient heart action. Digitalis requires from twenty-four to thirty-six hours to obtain its full effect, while the full effects of strychnia can be obtained in a few moments. This action of strychnia makes it by far the best heart stimulant to combat heart failure or to hold the heart together when dilated. As a result of much experience, we are convinced that in strychnia we have the most valuable and at the same time the most reliable heart stimulant known. The mistakes in the use of strychnia are two. First: The dose that is given is not enough to produce the stimulant effects. The tonic dose of strychnia is from 1-150 to 1-60 of a grain. The dose for its stimulant effect is from 1-30 to 1-25. The second mistake in the use of strychnia is expecting the drug to hold or continue its action for an indefinite time. In the use of strychnia, as soon as the heart begins to regain its strength, the dose should be reduced and some other heart tonic introduced to hold the effect obtained, as digitalis or the alkaloid caffeine. It is to be remembered that strychnia owes its action to the nervous system and that it can not be used for any great length of time without doing harm.

SPARTINE.— Is a heart remedy that acts very much like strychnia and at the same time acts somewhat upon the heart's muscle, which makes it a very valuable remedy when the left side of the heart is dilated. The extract of the suprarenal capsule in the form of the adrenal has a powerful and rapid action upon the arterial system, increasing the arterial tension and to an extent acting upon the heart muscle, increasing the force and

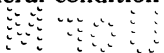
power of its contractions. In desperate cases of dilations of the heart it may be given in doses of 5 to 10 mms of the 1 to 1,000 solution, hypodermically.

ANEURISMS

"Aneurisms of the aorta are of two kinds: First, diffused; second, saccular. Saccular aneurisms involving the ascending portion of the arch of the aorta are apt to erode the sternum and penetrate the chest wall, while those of the transversed portion of the aorta or diffused aneurisms of the arch are more apt to extend into the chest and not to show themselves through the walls of the chest." We will consider first the signs of aneurisms and, second, the evidence of their locations.

The most important information regarding aneurisms is gained by inspection and palpitation. The patient should be in the recumbent position, with the head but slightly elevated, and so placed that the light will strike obliquely across the chest. The observer should cast his eye across the chest on a line with it, looking over the sternal region. In most cases of aneurism some abnormal pulsation will be seen to the right of the sternum or near the left scapular behind. If the sac be large, quite a surface of the sternal region or even behind this will be seen to pulsate; if the sac be small, but little or no pulsation will be observed. Not infrequently a distinct pulsation will be seen following the systole of the heart at the supersternal notch. Sometimes one can better feel the pulsation than see it. The pulsation is of an expansive character and has "an up-and-down motion." Occasionally the tumor is quite prominent and can be seen and felt as a hard, yet rather soft, enlargement on the surface. With the pulsation, one often feels a thrill, which is systolic in time, and again one will perceive by a light touch a dis-

tinct shock following the systolic thrill, the so-called "diastolic shock" which is not present when the sac walls become weakened. If the patient be placed in the sitting position and the observer stand behind him, resting the head of the patient upon his shoulder and with the head of the patient thrown somewhat back so as to stretch the trachea, and hold lightly the lower border of the cricoid cartilage, a distinct "tug," the tracheal tug, will be felt. This tug is an up-and-down movement and is felt during both inspiration and expiration. Other signs of aneurisms are: Unequal pupils, unequal radial pulse, pain in one arm, swelling of one arm or of one side of the head or neck. With the stethoscope, a systolic murmur or the diastolic shock may be heard. The diagnosis is based upon: First. The abnormal pulsations. Second. The presence of a tumor. Third. The thrill. Fourth. The diastolic shock. Fifth. The tracheal tug. Sixth. A systolic murmur. Seventh. Difference in the pupils or the pulse. As to the location of the aneurism, those of the ascending arch are apt to erode the sternum at the second right interspace. This erosion may be accompanied by pain, pulsation and other signs of the disease. Aneurism of the transversed portion, or diffused aneurisms, may show themselves by cough, unequal pupils and pulse, aphonia due to pressure upon the recurrent laryngeal nerve and the tracheal tug. Aneurisms of the descending portion of the aorta produce pain in the back and sometimes pulsation in the region of the scapula behind. The treatment of aneurisms of the aorta is very unsatisfactory. Ars. Iod., Kali. Iod., Murc. Sol., paying particular attention to rest and the general condition of the patient.



INDEX

	PAGE
Aconite	66, 90
Ammonia	90
Aneurism	99
Anasarca	37
Anatomy	7
Angina in aortic disease	6
pectoris	84
treatment	86
Aorta	10
Aortic area	20
second	21, 53
regurgitation	57
murmur	60
change in heart	57
pulse	59, 63
pulsation	60
impulse	58
apex	58
shock	60
symptoms	61
diagnosis	61, 70
compensation	61
relative leak	62
stenosis	22, 64
etiology	64
hypertrophy	64
pulse	65
symptoms	65
apex	64
murmur	64
diagnosis	65
treatment	66
Apex	20
in mitral regurgitation	32
in mitral stenosis	52
in aortic regurgitation	58
in aortic stenosis	64
Ars. Alb.	54, 90
Ars. Iod.	54, 90
Auricles	8
Auriculo-ventricular openings	9
Arteries, coronary	10
Bell	91
Bryonia	91
Cardiac cycle	14
Calomel treatment	45
Cactus	91
Chordæ tendinæ	9

	PAGE
Circulation	14
Codein	91
Caffeine	92
Columnæ carneæ	9
Coronary arteries	10
Cough	30, 37
Corrigan pulse	59
Collapsing pulse	59
Cratægus	91
Diuretine	46
Dropsy	37
Dyspnea	34, 37
Diastole	14
Diastolic murmur	60
Diagnosis	70
Digitaline	44
Dilatation	72
in mitral regurgitation	39
in mitral stenosis	54
in aortic regurgitation	62
in aortic stenosis
Digitalis	92
in mitral regurgitation	41
in mitral stenosis	55
in aortic disease	63, 66
Endocardium	9, 19
Endocarditis	74
Fibrous rings	10
Functional murmur	69
Hypertrophy	72
of right heart	32, 50
of left heart	58
Heart anatomy	7, 8
apex location	20, 52
diastole	14, 17
dilatation	72
fatty heart	81
fibers	11
fibrous rings	10
hypertrophy	32, 50, 58, 72
murmurs	21
nerves	11
physiology	14, 17
pause	14
systole	14, 17
sounds	17, 20, 32, 34
supports	13
stricture	10
Iodine	95